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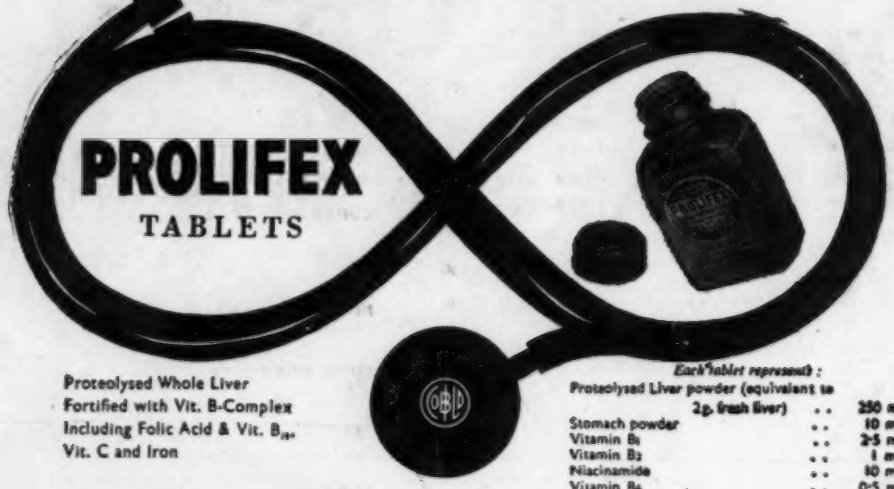

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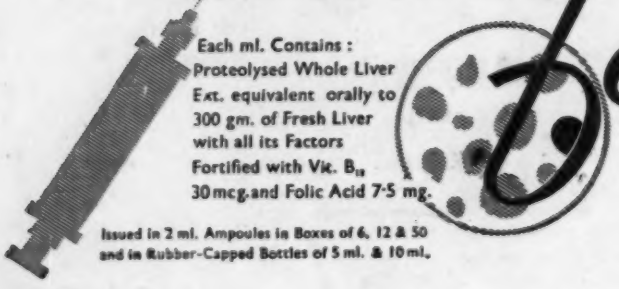

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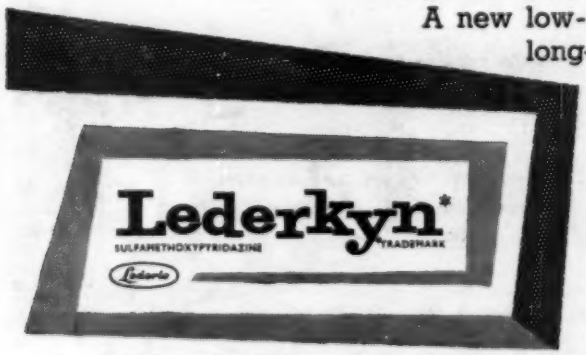

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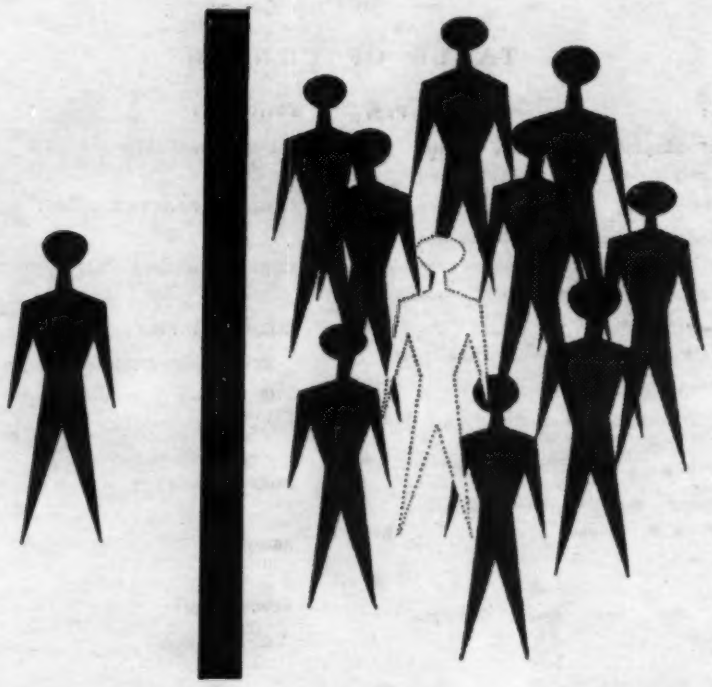

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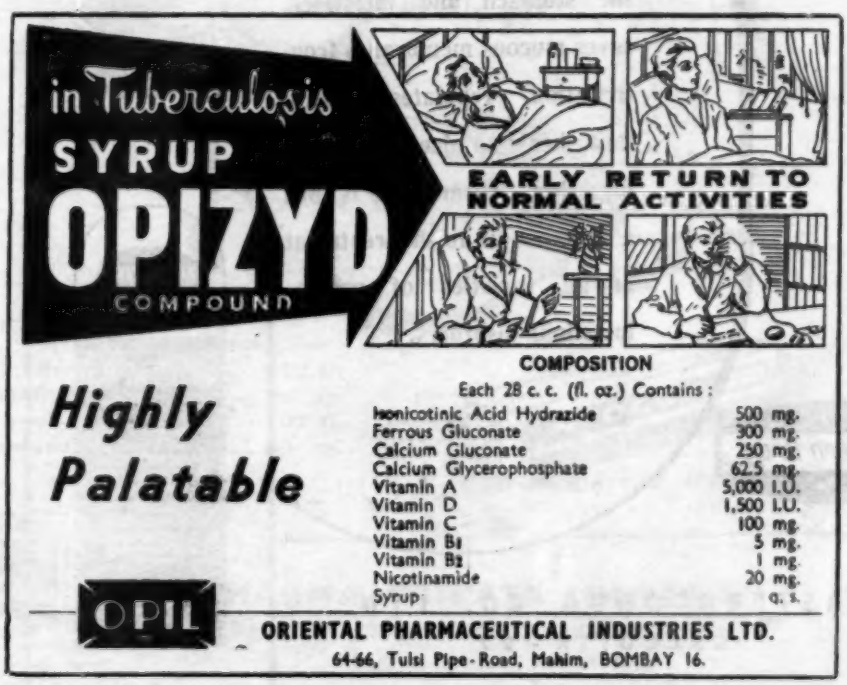

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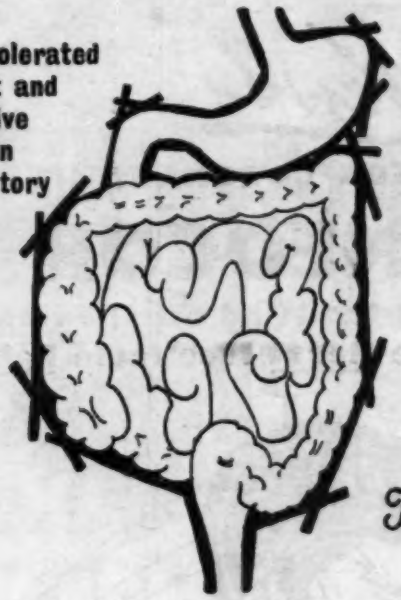

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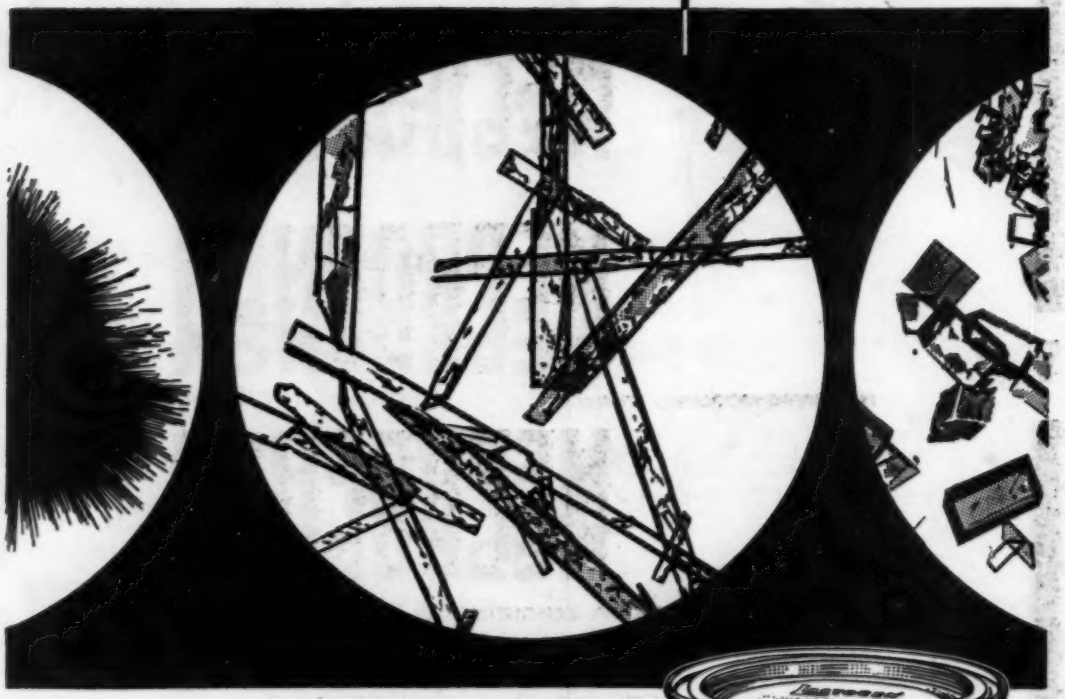

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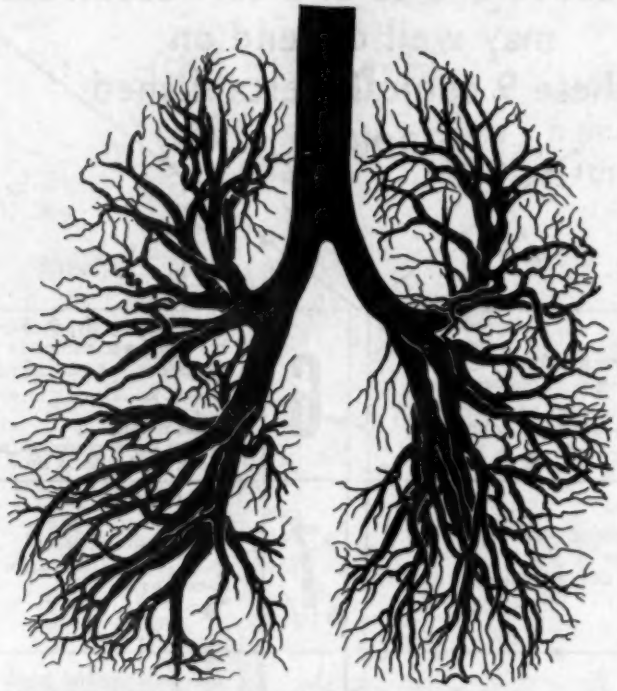

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1	initial therapy remarkably free from complications Allison, J. B., Sr., and Allison, J. B., Jr.: Monographs on Therapy 3:99 (October) 1958.
2	continuing therapy-maintenance doses are low Feinberg, S. M., *et al.*: J.A.M.A. *167*: 58 (May 3) 1958.
3	no sodium or water retention—low salt diet not necessary Hartung, E. F.: J.A.M.A. *167*: 973 (June 21) 1958.
4	absence of edema Council on Drugs: J.A.M.A. *169*: 257 (January 17) 1959.
5	less likely to create electrolyte disturbance Bongiovanni, A. M., *et al.*: J. Pediat. *53*: 3 (July) 1958.
6	no secondary hypertension — no significant change in pulse, respiration, or blood pressure Shelley, W. B., *et al.*: J.A.M.A. *167*: 959 (June 21) 1958. Berntsen, C. A., Jr., *et al.*, New York Rheumatism Association, Annual Meeting, New York, April 9, 1959.
7	no excessive appetite Council on Drugs: J.A.M.A. *169*: 257 (January 17) 1959.
8	without unnatural psychic stimulation—does not stimulate and rarely depresses the mood Shelley, W. B., *et al.*: J.A.M.A. *167*: 959 (June 21) 1958. Council on Drugs: J.A.M.A. *169*: 257 (January 17) 1959.
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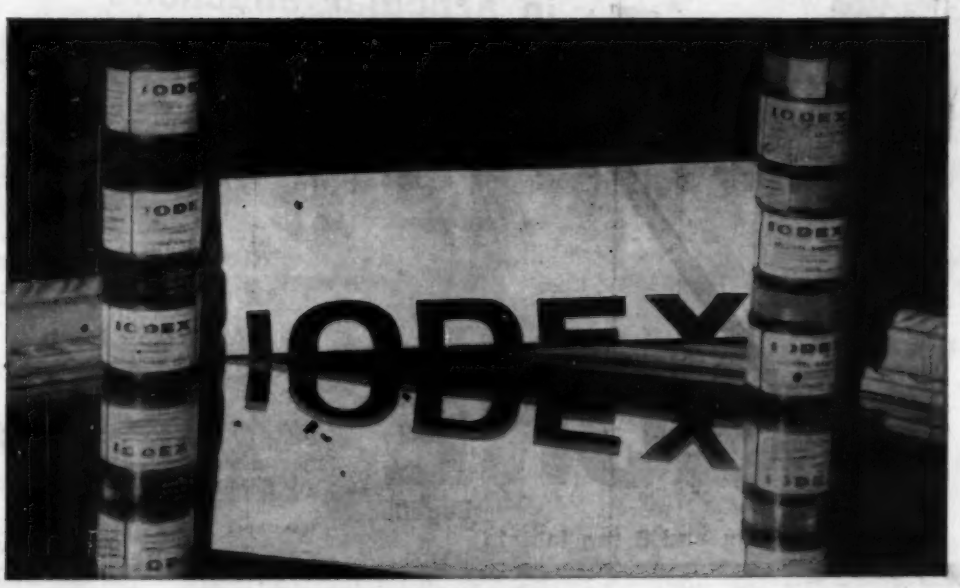

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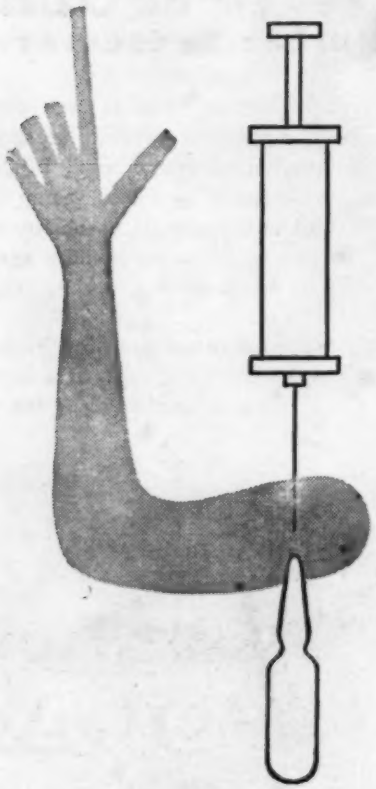

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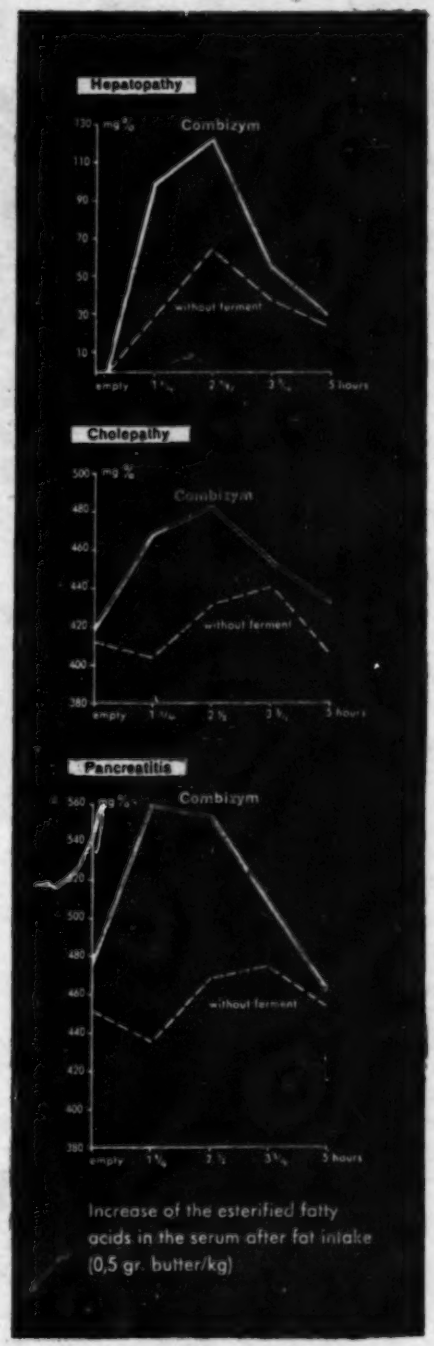

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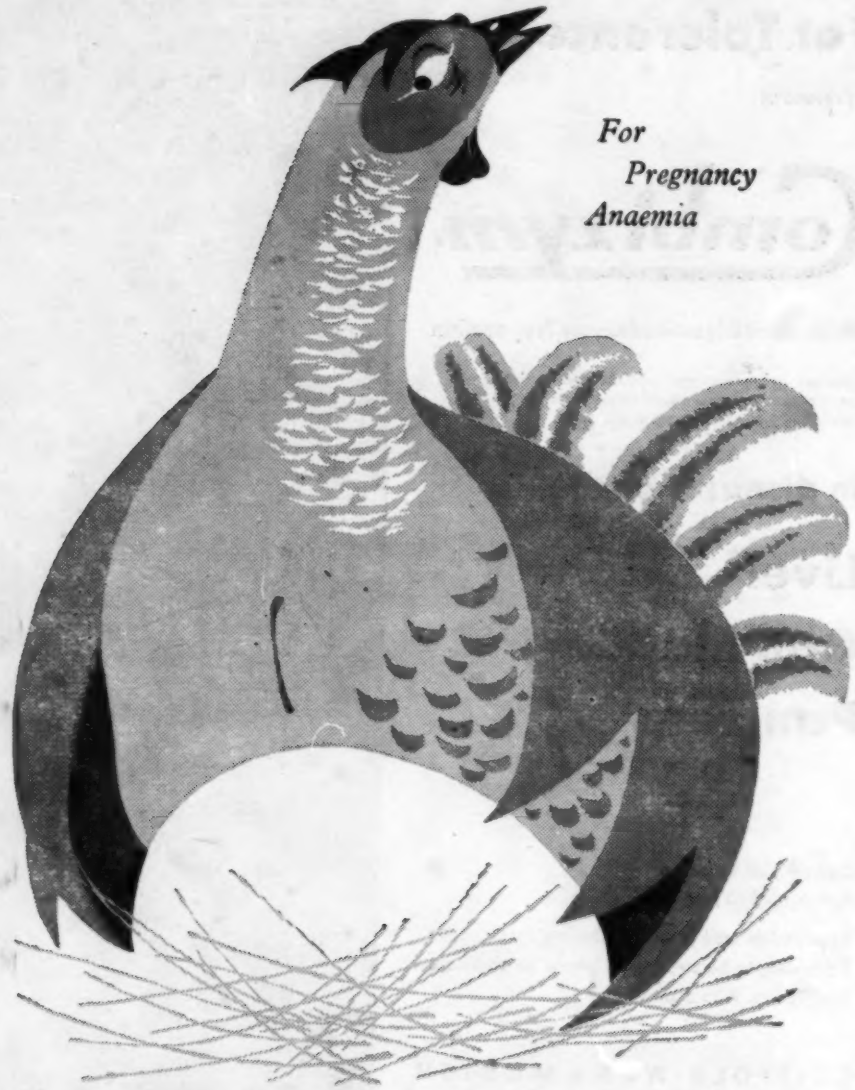

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Anaemia

TONOFERON ***MAXIMUM IRON IN MINIMUM DOSE***

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ORIGINAL ARTICLES

MANAGEMENT OF IDIOPATHIC ULCERATIVE COLITIS*

R. N. CHAUDHURI, M.B., F.R.C.P. (EDIN.), T.D.D. (WALES), F.N.I.

M. K. CHHETRI, M.D. (CAL.), M.R.C.P. (LOND.)†

AND

S. B. CHATTERJEE, M.B.B.S. (CAL.)

School of Tropical Medicine, Calcutta

Idiopathic ulcerative colitis is a disease of obscure aetiology, possibly related to multiple factors and characterised by inflammation and ulceration of the mucosa and submucosa of the large gut, often appearing first in the rectosigmoid region and then spreading towards the caecum, with subsequent reparative fibrotic changes. The common manifestations are bloody diarrhoea containing pus and mucus with or without fever, and their after-effects, viz., dehydration, anaemia, hypoproteinaemia and loss of weight. The disease tends to run a chronic course with remissions and relapses, and occasionally it may be fulminant in type. Management is complex, requiring a broad approach of physical and emotional rest, diet and other allied measures. The availability of chemotherapeutic agents, steroid hormones, facilities for correction of fluid and electrolytic imbalance and modern surgery has considerably changed the outlook of such patients in recent times.

The present paper deals with our experiences with the management of 31 cases, all Indians, of ulcerative colitis admitted to the hospital attached to the School.

There were 21 males and 10 females, the age varying from 9 to 51 years, the average being 28 years. The disease was diagnosed clinically as well as by proctosigmoidoscopy and barium enema skiagraphy, other diseases, such as specific dysenteries having been excluded by repeated stool examinations, both microscopically and culturally. The patients were admitted in different phases of the illness, the duration varying from 1½ months to 16 years. They were broadly subdivided into three groups, viz., acute, subacute and chronic (Table 1).

In the absence of any specific drug for ulcerative colitis the treatment in these cases was mainly symptomatic, supportive and corrective and included the following measures:

1. *Rest*—Bed-rest was insisted upon in the acute and subacute phases with active symptoms. Otherwise, restricted physical activities were permitted, depending on the severity of the illness. Emotional rest was attempted by reassuring the patient, trying to understand his psychological problems sympathetically and by obtaining mild sedation with phenobarbitone (gr. ½ to 1, thrice daily). In this respect, hospitalisation was of distinct value, helping removal of the patient from his own environmental influence and establishing a close doctor-patient relationship and providing facilities for a more intensive treatment.

2. *Diet*—In the acute phase of the illness, the diet was mainly liquid and bland and consisted of

* Based on a symposium held under the auspices of the Indian Medical Association, Calcutta Branch.
† Now visiting physician, Seth Sukhlal Karnani Memorial Hospital, Calcutta.

TABLE 1—SHOWING AGE, SEX, CLASSIFICATION OF CASES, TREATMENT AND RESULT IN 31 CASES

Sl. No.	Initials (Age/Sex)	Stage of illness	Sodium sulphate	Sulpha-guanidine	Antibiotics	Steroids	Result and comments
1.	SKB (32/M)	Acute	0	0	0	Cortisone	Rapid and marked improvement
2.	BM (20/F)	Acute fulminant	0	0	Chlorostrep (oral)	Cortisone, later hydrocortisone	No improvement. Subjected to ileostomy but died
3.	AB (13/F)	Acute	+	0	0	Cortisone	Partial improvement with sodium sulphate, slightly more with cortisone
4.	CAL (44/M)	Subacute	0	0	Terramycin (oral)	Cortisone	No improvement
5.	SRS (30/F)	Subacute	0	0	0	Cortisone	Marked improvement
6.	ARD (51/M)	Chronic	0	0	0	Cortisone	Partial improvement
7.	HS (35/F)	Acute	0	0	0	Cortisone	Partial improvement
8.	MRB (35/F)	Chronic	+	0	0	Prednisolone	Slight improvement with prednisolone, but later marked improvement with sodium sulphate
9.	AFMH (32/M)	Acute	+	0	0	Prednisolone (in relapse)	Dramatic remission with only sodium sulphate. Relapse partially improved even with prednisolone
10.	RDG (20/F)	Chronic	+	0	0	Prednisolone	Only partial improvement
11.	PKA (16/M)	Chronic	0	0	Streptomycin injections	Cortisone	No improvement
12.	RBD (31/M)	Subacute	0	0	0	Prednisolone	Marked improvement.
13.	LK (9/F)	Acute	0	0	0	Prednisolone	Marked improvement.
14.	BDJ (45/M)	Chronic	0	0	0	Prednisolone	Marked improvement.
15.	SPS (28/M)	Subacute	0	0	Penicillin & streptomycin injections	Triamcinolone	Marked improvement.
16.	MD (26/F)	Subacute	+	0	Streptomycin injections	Triamcinolone	Marked improvement.
17.	NVG (35/M)	Early subacute	0	0	0	Triamcinolone; hydrocortisone (rectal drip)	No improvement with triamcinolone (oral) but improved with hydrocortisone (rectal drip)
18.	BBD (26/M)	Chronic	0	0	0	Triamcinolone; hydrocortisone (rectal drip)	No effect with rectal drip, but later marked improvement with triamcinolone
19.	NCR (26/M)	Acute	0	0	0	Prednisolone	Partial improvement; later subjected to ileostomy but died

TABLE 1—*Contd.*

Sl. No.	Initials (Age/Sex)		Stage of illness	Sodium sulphate	Sulphaguanidine	Antibiotics	Steroids	Result and comments
20.	LM	(31/F)	Subacute	+	0	0	Prednisolone	Marked improvement. Developed transient oedema which soon subsided with salt-restriction
21.	SRG	(32/M)	Subacute	+	0	0	Dexamethasone	Slight improvement
22.	HM	(30/M)	Acute	0	0	0	Prednisolone	Partial improvement, but soon relapsed even on maintenance dose
23.	SCM	(26/M)	Acute	+	+	0	Dexamethasone	Responded to first course of sulphaguanidine, but not later during relapse. Responded partially to dexamethasone although loose motions continued
24.	KCM	(25/M)	Chronic	0	Succinyl sulphathiazole	0	Prednisolone	Marked improvement
25.	AS	(34/M)	Chronic	+	+	Penicillin & streptomycin injections	Dexamethasone	Stool became formed, but blood and pus persisted which further lessened with dexamethasone & penicillin-streptomycin
26.	JLD	(22/M)	Acute	+	+	Penicillin & streptomycin injections	Triamcinolone	Partial improvement with penicillin, streptomycin and sulphaguanidine. Triamcinolone produced massive haemorrhage
27.	IA	(32/F)	Clinical remission	0	0	0	0	Improved with diet, rest, antispasmodics (belladonna) and sedatives
28.	DNB	(51/M)	Subacute	+	+	Penicillin & streptomycin injections	0	Marked improvement
29.	SB	(15/M)	Subacute	+	+	Penicillin & streptomycin injections	0	Marked improvement
30.	AB	(22/M)	Subacute	+	+ (incomplete)	Streptomycin (oral)	0	Marked improvement with streptomycin. Had allergic skin reaction with sulphaguanidine
31.	BNG	(18/M)	Subacute	+	+	0	0	Slight improvement with sodium sulphate, marked improvement with sulphaguanidine

skimmed or butter-milk, soup and fruit juice. Adequate fluid intake (3 to 4 pints a day) was assured and intravenous glucose also supplemented the intake, wherever necessary. In severe cases, water and electrolyte deficiencies were corrected by intravenous infusion of normal saline with or without 5 per cent glucose and oral administration of potassium chloride, 2g., a day. Boiled fish and dry toasts were added after 3 or 4 days with improvement of the appetite and the character and consistency of the stool. The diet was gradually increased, avoiding roughages and spices. In chronic or subacute phase, the diet consisted of high calories, high protein, low carbohydrate and fat diet, nutrition being of major importance in ulcerative colitis. Sodium and potassium salts were administered according to necessity (e.g., frequency of stools, asthenia, etc.). Vitamins, particularly vitamin C and B-complex, were supplemented to the diet of all the patients.

3. *Blood transfusion*—Blood transfusions were given to patients having considerable loss of blood in the stool. Ten patients in this series required one or more transfusions during the course of illness while in the hospital.

Incidentally haematinics were helpful after the bowel condition had subsided. Iron, folic acid and/or liver extract were given to the majority of the patients according to the nature and degree of anaemia. Sometimes their administration was preceded by blood transfusions. Some patients did not tolerate oral iron preparation in full doses.

4. *Astringents*—For control of diarrhoea bismuth and kaolin were given to almost all patients before, and to a few after admission. Two patients were treated with silver nitrate (1 in 10,000) bowel wash, which was found to be of no value. For the purpose of night's rest, administration of bismuth and ipecac powder or a dose of codeine for a night or two was helpful in a few patients.

Bael fruit (*Aegle marmelos*) has long been reputed in this country for its mild astringent, lubricant and demulcent actions on the bowel. A preparation of it in powder form (pulv. marmelos), 4 to 6 teaspoonfuls a day, was tried on a few patients for persistent loose mucoid stools. Two had marked improvement, two had only slight improvement, and the other one had none.

5. *Antispasmodics*—Belladonna was administered to several patients but it did not seem to be of any significant value except for occasional relief of colicky pain in 2 or 3 patients.

6. *Antibiotics*—Most patients had received broad-spectrum antibiotics (particularly of the tetracycline group) before admission, but in some of them the condition had worsened. In the hospital, chloromycetin and streptomycin (chlorostrep) in one patient and terramycin in another, by the oral route, were administered without any benefit. A third patient treated with streptomycin tablet orally showed partial improvement. Two other patients had parenteral streptomycin, 1 g. daily for 10 days, without any effect ; the result was similar in the case of a patient treated with both streptomycin and penicillin parenterally. Five patients were treated with oral sulphaguanidine with a starting dose of 12 tablets followed by 6 tablets every 6 hours for 10 to 14 days, two patients along with parenteral penicillin and streptomycin. The last two patients were in the subacute phase of illness and had almost complete remission of symptoms. In other 3 patients also, there was marked improvement with formed stool and lessening of blood. But one patient in the acute phase had rapid exacerbation soon after discontinuation of the treatment and did not respond any more to a second course of sulphaguanidine. One patient treated with phthalyl-sulphathiazole (thalazole) showed partial improvement of chronic illness.

7. *Mild saline laxative*—Sodium sulphate, 2 teaspoonfuls in water, was given twice a day for 2 days and then once in the morning on an empty stomach to 18 patients with foul-smelling stools and fever. The object was to drain out the necrotic or putrefying materials in the intestinal lumen, and thus to check the flourishing bacteria. Five patients showed a striking improvement, and six others only partial improvement, while the remaining 7 remained practically unaffected.

8. *Steroids*—Altogether 26 patients had steroids. Eight of them received cortisone tablets, starting with 75 mg. a day in divided doses (children, half the dosage) for 7 to 10 days, 50 mg. a day for another week and subsequently 25 mg. a day as maintenance dose for 2 to 4 weeks. Ten were given prednisolone tablets, 20 mg. a day in divided doses for 7 days—being tailed off every week till a maintenance dose of 2·5 to 5 mg. a day was reached. Triamcinolone was administered with the initial dose varying from 8 to 16 mg. a day in 5 patients with gradual tapering off as usual. Dexamethasone was given to three patients with the initial dose of 2 and 3 mg. a day in divided doses in a similar manner. Hydrocortisone hemisuccinate sodium (100 mg.) was administered to 2 patients daily as retention enema for 4 and 10 days respectively. A daily check of the blood pressure, restriction of salt in the diet and measurement of body-weight were done. No side-effects were seen, except in a case with mild diabetes

where the blood sugar had shot up transiently and in another patient having cortisone who had developed temporary oedema. These effects promptly subsided, however, with cessation of the drug and institution of salt-free diet respectively.

The improvement was rapid and striking in 7 cases, being evident from 48 hours of the steroid therapy. At first, the temperature started settling down and was followed by improvement in the number and consistency of the stools and reduction in the amount of blood passed. Subseqently, the appetite increased with a sense of well-being. Improvement of anaemia and gain in weight were noted later on.

Ten patients showed partial improvement as regards blood in the stools, fever and a sense of well-being. All but one were in the chronic phase of the disease and except two all had the trouble of more than one year's duration. In another patient in the acute phase of the disease with pseudopolyposis of the gut, the subjective symptoms improved and the fever was controlled but the diarrhoea persisted. He was subsequently subjected to surgery and died. Two other patients with pseudopolyposis had only partial improvement.

Two patients were given slow rectal drip with hydrocortisone hemisuccinate, one in the subacute and the other in the chronic phase of the illness. The former improved markedly only with 4 rectal drips, but the latter did not ; subsequently he had remarkable and lasting improvement with triamcinolone given orally.

In 4 patients, no improvement was observed. One of them came in the acute fulminant phase of the illness and was subsequently subjected to ileostomy after cortisone followed by hydrocortisone therapy had failed, but surgery was also ineffective in saving her. The remaining three were in the chronic phase of the illness. Massive intestinal haemorrhage was preciptated with triamcinolone in one patient for which the drug had to be stopped.

Discussion

Idiopathic ulcerative colitis is a disease of undetermined aetiology with a mysterious pathogenesis, non-specific histological picture, and of unpredictable course. As remissions and relapses are common, it is hard to assess the efficacy of a particular form of therapy unless the patients are followed up for a long time. The present series is a small one ; nevertheless, it enables us to make certain observations with regard to management.

Hospitalisation of the patient is desirable whenever possible, as this provides a change in the environment and a close relationship between the doctor and the patient. Rest—physical, emotional and gastro-intestinal—definitely improves the clinical state. Astringents and adsorbents are practically without any effect on the bloody diarrhoea. Bowel wash is not usually helpful. A mild saline laxative, such as sodium sulphate in small doses, is definitely helpful to ameliorate the clinical symptoms, viz., fever and offensive stools, perhaps by removing necrotic and putrefying materials from the lumen of the bowel. It is preferable to astringents. Occasionally preparations of *bael* (*Aegle marmelos*) are helpful especially in chronic stages. Broad-spectrum antibiotics, which most of the patients in this series received before coming under our observation, have nothing very much to commend. They may aggravate the condition by inducing post-antibiotic colitis. There is also the danger of developing resistant strains of bacteria and overwhelming monilia infections. Such antibiotics should be reserved for patients with severe toxaemia and other complications. Sulphaguanidine or a similar preparation by mouth may be tried especially in mild or moderately severe ulcerative colitis and should be continued for 10 days or more. A special preparation, namely, salicylazosulphapyridine (asulfidine) has been particularly recommended for ulcerative colitis, but it was not available to us. The substance is an azo compound of salicylic acid and sulphapyridine and is supposed to have an affinity for connective tissue. Unfortunately, it is said to cause a high incidence of skin eruptions. Blood transfusion is a valuable adjunct to therapy, especially for anaemia which is principally due to blood loss and infection. In severe cases fluids and electrolyte depletion may be marked and require parenteral replacement. Potassium should be supplied in such cases.

Steroid therapy induces clinical remission in a larger percentage of cases, that is, in over 40 per cent, as compared to 13 to 17 per cent in the control series, particularly in the first attack and in the relapse phase of the illness (Truelove and Witts, 1954, 1955). This is well illustrated by the dramatic response of seven patients in this series with rapid control of fever and bloody fluxes, while the conservative treatment had failed. This response to treatment has a tremendous rejuvenating effect on the psychological set up of these patients. Incidentally, however, in a smaller series of cases reported previously from this institution (Chaudhuri, 1954), the result of treatment with ACTH and cortisone was not found to be

encouraging. More favourable results were, however, obtained subsequently (Chaudhuri, 1957-58, and Chatterjee, 1957). The drug has been used in varying doses by different workers, viz., cortisone 100 to 300 mg. daily (Truelove and Witts, 1954, 1955), prednisone or prednisolone 40 to 80 mg. daily (Kramer, 1958). In this series, the dosage used was relatively smaller and yet the results had been fairly satisfactory with practically no untoward effect or complications known to supervene on large doses or on prolonged therapy, except in one case in whom massive haemorrhage was precipitated. Truelove and Witts (1959) in a series of 169 cases of ulcerative colitis treated with corticotrophin (84 cases) and cortisone (85 cases) with the usual large doses, reported complications in 35 and 24 cases respectively and they further stated that the result with cortisone was practically identical with their earlier series (1954) though the dosage was on an average, twice as large. The relapse rate after steroid therapy, though varying according to different workers, is considerable, e.g., 70 to 80 per cent (Kramer, 1958) and about 50 per cent (Maltby *et al*, 1956). Of the 34 patients who had short-term hormone treatment, only 12 remained in remission at the end of 2 years (Elliott and Giansiracusa, 1954). Here no attempt was made to study the effects of steroids on the suppression of relapses on maintenance therapy or the relapse rate after therapy.

The response to steroid therapy was poor in chronic phases of the illness. Thus 10 patients had partial response and 4 did not respond at all —the majority being in the chronic phase. One of them was in the acute fulminant phase, who did not respond at all to steroid therapy. Hydrocortisone hemisuccinate sodium in a nightly dose of 100 mg. in 100 to 120 ml. of saline for 2 to 3 weeks in slow rectal drip has been advocated by several workers (Truelove, 1956, 1957, 1958 ; Nabarro *et al*, 1957 ; Watkinson, 1958). This was tried in one patient in the early phase of the disease ; he responded well, and stools became normal after only 4 consecutive nightly administrations. Another case was practically uninfluenced by administration of rectal hydrocortisone drip even for 10 consecutive nights. He, however, responded well to oral triamcinolone in a dose of 16 mg. initially. Local administration of other preparations like prednisone or prednisolone has also been tried with good results by some workers (Patterson and McGivney, 1959 ; Matts, 1960), but none of our cases was so treated. These experiments indicate that steroids are most potent when given to cases with superficially involved colon rather than to cases with chronically thickened and fibrotic colon.

Two patients, who had received steroid but did not respond, where ultimately treated surgically but both succumbed. It is stated by some authors (Bockus *et al*, 1958) that steroid therapy (when it fails) makes the patient a bad surgical risk (21 per cent mortality compared to 7 per cent mortality in patients not previously on steroid therapy). Hayes and Kushlan (1956) also had similar experience.

Summary

1. Observations on 31 cases of idiopathic ulcerative colitis with special reference to management are presented and discussed. Since no specific aetiological agent has been found, treatment is largely supportive and symptomatic.

2. Bed-rest, preferably in a hospital with sedatives like phenobarbitone, is the basis of general management.

3. The diet is restricted for a few days depending on the condition of the patient. It is worked up to the usual colitis diet, fats and carbohydrates being restricted. Milk may be skimmed or replaced by butter milk. Nutrition being an important factor in ulcerative colitis, the diet should be adequate, palatable and non-monotonous as far as possible, avoiding commonly known offenders, such as curries, irritants and coarse vegetables.

4. Fluids and potassium chloride are administered to correct dehydration, salt depletion and acidosis in cases with persistent diarrhoea and weakness. Vitamins especially vitamin B-complex and C, are also helpful. If indicated, intravenous glucose saline and blood transfusion should be given.

5. Mild saline laxatives are given in the form of sodium sulphate, two teaspoonfuls in water two or three times a day for two to three days (unless the patient is very dehydrated) followed by a small morning dose for a few days. They are more useful than astringents and antispasmodics.

6. After a few days' trial with sodium sulphate, the patient is put on sulphaguanidine, 12 tablets (6 g.) stat and then 6 tablets (3 g.) 6 hourly for at least 10 days, besides sedation and colitis diet.

7. Along with sulphaguanidine, the patient may be given streptomycin and penicillin injections to combat secondary infections.

8. Thereafter, the case is carefully assessed :

(a) If improved, he is patiently kept on rest, diet with minor restrictions and mild sedative. Purgatives are to be avoided as far as possible.

(b) In the absence of satisfactory response, the steroids are given. This therapy is indicated in patients (i) with active phases of illness, including acute exacerbations of chronic ulcerative colitis, (ii) who have failed to respond to other medical measures preferably in a hospital, (iii) with systemic complications, viz., arthritis, erythema nodosum, etc. The steroid hormone, although in no sense curative, helps to tide over the hazardous state of illness in a large proportion of cases. Unfortunately, recurrences are not uncommon, when the problem may be difficult.

9. Chronic cases, especially when medical treatment has failed, may require surgery. Steroids should not be continued too long, as such cases become poor surgical risks. Besides, side-effects may supervene.

10. When cases with acute fulminant colitis do not respond within a few weeks to medical measures including steroids therapy and transfusions surgical treatment should be considered without delay, as the major mortality, whether medical or surgical, falls in this group.

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BIOCHEMICAL STUDIES OF ESCHERICHIA COLI ISOLATED FROM CASES OF URINARY TRACT INFECTION AND THEIR SENSITIVITY TO SULPHA DRUGS AND ANTIBIOTICS

B. S. DARBARI, B.SC., M.D.

Lecturer in Pathology

AND

B. P. SAXENA, M.D., PH.D. (LOND.)

Professor of Pathology and Bacteriology

G. R. Medical College, Gwalior

Escherichia coli infection of the urinary tract is a common condition. In trying to find out the presence, if any, of special strains of *E. coli* proving more pathogenic than others and producing urinary tract infections, a detailed biochemical study of the organism was undertaken. As many of the cases were chronic and did not respond to one or more of the sulpha drugs and/or antibiotics, the isolated strains of *E. coli* were subjected to drug sensitivity tests.

Material and Methods

The investigations were carried out on 89 strains of *E. coli* isolated from 83 patients, 29 male and 55 female, suffering from urinary tract infections in the J. A. Group of Hospitals, Gwalior.

Catheterised samples of urine from female patients and either catheterised or midstream specimens of urine from male patients were collected aseptically. A part of the centrifuged urinary deposit was inoculated on MacConkey's medium and the rest was examined for pus cells and micro-organisms. After 24 hours, pink colonies of gram-negative bacilli from MacConkey's medium were selected and subcultured in peptone water. This was used for further subcultures.

The following 12 sugars were used for fermentation reactions :

Glucose ; lactose ; maltose ; mannitol ; adonitol ; arabinose ; dulcitol ; inulin ; inositol ; xylose ; sucrose and salicin.

For methyl-red reaction, citrate utilisation, indole production and Eijman reaction techniques described by Mackie and McCartney (1953) were used. For Voges-Proskauer reaction, Barritt's (1936) modification was used. For gelatin-liquefaction, Wilson and Miles' (1956) technique was used. Haemolytic activity was tested as sug-

gested by Dudgeon *et al* (1921), drug sensitivity tests were performed by disc method using the technique of Lund (1949) as modified by Fairbrother and Martyn (1951) and Gupta and Chakravarti (1954). 2·5 per cent agar in papain digest both plates (6-8 mm. thick) were inoculated with 1 in 1000 dilution of 24-hour broth culture in normal saline. Inoculation was done by pouring about 2 ml. of the diluted culture and pipetting off the excess by tilting the plate. After allowing it to stand at 37° C for 15 minutes, the plates were divided into six radial zones with a glass pencil. In the centre of each zones was placed a sterilised filter paper (Whatman No. 1) disc of 6·2 mm. diameter. One drop of each of the following drugs in strength mentioned was placed on the respective discs with the help of a No. 1 gauze needle fitted to a syringe (Table 1): Sulphadiazine, sulphamerazine and streptomycin each of 1 per cent solution and chloromycetin, achromycin and terramycin each of 0·25 per cent solution.

After 24 hours' incubation at 37° C the diameter of the zone of inhibition was measured in millimeters. The degree of drug sensitivity was noted as follows:

The strain was considered resistant (0) when the diameter of the zone of inhibition measured upto 5 mm., slightly sensitive (+) when it measured between 6 and 10 mm., moderately sensitive (++) when between 11 and 15 mm. and highly sensitive (+.++) when the measurement was 16 mm. and above.

The solutions of sulpha drugs and antibiotics were stored at 4°C and used only for one week after preparation.

Observations

(A) *Biochemical studies*—The strains of *E. coli*, isolated from cases of urinary tract infections and grouped according to Wilson's (1935) classifiction are shown in Table 1.

Table 1—Showing Classification of Strains of *E. coli*

Microorganisms	Type	No. of strains
Bact. coli (*E. coli*) ...	I	28
	II	2
Intermediate	I	2
	II	11
Bact. aerogenes (Aerobacter) ...	I	1
	II	3
Regular strains	I	9
	II	1
	III	1
Total ...		58

31 strains could not be categorised according to Wilson's classification and have been grouped as authors' irregular strains. These strains, with their biochemical properties, are shown in Table 2.

Table 2—Showing Biochemical Properties of the Authors' Irregular Strains

Authors' irregular strains	Methyl red reaction	Voges-Proskauer reaction	Citrate utilisation	Indol production	Eijkman reaction	Gelatin liquefaction	No. of strains
I ...	−	−	+	+	+	−	10
II ...	−	−	−	+	+	−	7
III ...	+	−	+	+	+	−	7
IV ...	−	−	−	+	−	−	3
V ...	−	+	+	+	−	+	2
VI ...	−	−	+	−	+	−	1
VII ...	−	+	+	+	+	−	1
Total ...							31

(B) *Sugar fermentation reactions*—Results of these tests are shown in Table 3.

Table 3—Showing Results of Sugar Fermentation Reactions

Serial No.	Sugars used in the test	No. of strains used in the test	No. of strains producing acid and gas	No. of strains producing acid only	No. of strains not fermenting the sugar
1.	Lactose ...	89	86	1	2
2.	Glucose ...	89	88	1	0
3.	Maltose ...	89	88	1	0
4.	Mannitol ...	89	88	1	0
5.	Arabinose ...	79	78	1	0
6.	Xylose ...	79	71	5	3
7.	Dulcitol ...	79	49	0	30
8.	Inositol ...	79	6	12	61
9.	Adonitol ...	79	4	5	70
10.	Inulin ...	79	0	4	75
11.	Sucrose ...	82	48	12	22
12.	Salicin ...	82	52	12	18

(C) *Haemolytic tests*—Results of haemolytic tests are shown in Table 4.

Table 4—Showing Results of Haemolytic Tests

Type of microorganism	Number
Haemolytic strains	35
Non-haemolytic strains	54
Total	89

(D) *Drug sensitivity tests*—76 of the isolated strains were put to drug sensitivity tests against

the commonly used sulpha drugs and antibiotics. These results are summarised in Table 5.

TABLE 5—SHOWING RESULT OF DRUG SENSITIVITY TESTS

Drugs used	No. of strains				
	Resistant (0)	Slightly sensitive (+)	Moderately sensitive (++)	Highly sensitive (+++)	Total
Sulphadiazine	31	28	12	5	76
Sulphamerazine	41	28	5	2	76
Streptomycin	10	25	23	18	76
Chloromycetin	13	19	20	24	76
Achromycin	7	20	13	36	76
Terramycin	12	17	10	37	76

DISCUSSION

Wilson's classification of the coliform bacilli—Out of 89 isolated strains of *E. coli* 58 strains could be fitted in Wilson's classification of coliform bacilli, while the rest which could not be so classified, have been put as authors' irregular strains.

According to the biochemical tests done on the lines of Wilson's classification, 28 strains of the *E. coli* isolated were found to be of faecal variety and were typical coliform bacilli probably from the gut. The rest of the strains belonged to a typical or non-faecal group. This means that either the latter have changed to a typical form, becoming more pathogenic and thereby getting an access to the urinary tract and producing infection there, or that these organisms are atypical strains coming from outside as contaminants and producing the infection.

Sugar fermentation reactions—Out of 89 strains put to test, 86 fermented lactose with acid production, while 1 produced only acid and 2 did not ferment lactose and belonged to the group of so-called 'para-colon' bacilli. One strain was of *E. coli* anaerogenous type. All the strains fermented glocuse, maltose and mannitol and most of them arabinose, xylose and a fair number fermented dulcitol, while very few fermented adonitol, inositol and inulin.

As suggested by Winslow *et al* (1919), Bray (1945) and Wilson and Miles (1955), 82 of the isolated strains were classified on their property to ferment sucrose and salicin (Table 6).

Haemolytic activity—Out of 89 strains isolated the authors found only 35 (40·6 per cent) producing haemolysis. Almost similar were the findings of Dudgeon *et al* (1921) who found that 42 per cent of their strains, isolated from cases of urinary tract infections were haemolytic.

TABLE 6—SHOWING ORGANISMS WHICH ARE SUCROSE AND SODIUM FERMENTERS

Organisms	Sucrose	Salicin	No. of strains
Var neapolitanum	+	+	51
Var communins	+	−	9
Var commune	−	+	13
Var acidi lactici	−	−	9

On the whole it appears that haemolytic property of *E. coli* is related to its pathogenicity but does not form an all important landmark in pronouncing the strains as not pathogenic, for even non-haemolytic strains have proved to be pathogenic.

Drug sensitivity tests—Out of 89 strains, 76 were put to drug sensitivity tests.

Majority of the strains of *E. coli* were resistant to sulpha drugs and 31 strains (40·7 per cent) were resistant to sulphadiazine and 41 strains (53·9 per cent) to sulphamerazine. This high resistance against sulpha drugs has also been reported by Gupta and Chakravarti (1954) using sulphathiazole as the drug. As suggested by these authors this resistance to sulpha drugs may be due to their indiscriminate use.

As against sulpha drugs, there were comparatively much fewer number of strains which were resistant to antibiotics, e.g., only 10 strains (13·1 per cent) were resistant to streptomycin, 13 (17·1 per cent) to chloromycetin, 7 (9·2 per cent) to achromycin and 12 (15·7 per cent) to terramycin. Terramycin and achromycin have proved to be the better choice among antibiotics, as compared to chloromycetin and streptomycin. The percentage of sensitive strains to antibiotics as found in the present study is almost the same as that reported by Gupta and Chakravarti (1954), except the strains tested with achromycin.

The high percentage of sensitivity to these antibiotics may be due to the fact that patients are relatively less frequently treated with these antibiotics as compared to sulpha drugs.

SUMMARY

89 strains of *Escherichia coli* isolated from 83 patients have been investigated. 58 of these strains could be fitted in Wilson's classification of coliform bacilli. The remaining 31 isolated strains have been grouped as the authors' 'irregular strains'. Fermentation reactions with 12 different

sugars were noted. Haemolytic property of *E. coli* was investigated. 35 strains were found to be haemolytic while 54 strains were non-haemolytic. Drug sensitivity tests *in vitro* were performed against sulpha drugs and antibiotics. Majority of the strains were found to be resistant to sulpha drugs. Amongst the antibiotics achromycin and terramycin were found to be better tolerated than streptomycin and chloromycetin.

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A NEW LINE IN THE TREATMENT OF TETANUS

B. N. SINHA, M.D., M.R.C.P. (EDIN.), F.R.F.P.S. (GLAS.)

Department of Medicine
Darbhanga Medical College, Laheriasarai

Tetanus continues to take a heavy toll of life in eastern countries. Its high incidence is largely due to the poor socio-economic standards in these countries, social and religious prejudices being also contributory factors. Coupled with these is the prohibitive cost of modern methods of treatment.

The treatment of tetanus has evolved through ages and even though no specific cure is yet available, many potentially valuable drugs and techniques are now employed with advantage. The newer trends hold that therapy should be aimed at limiting the progress of the disease and preventing further elaboration and fixation of toxins. More specifically the therapy is designed to overcome progressive and persistent rigidity and ensure adequate air entry. The importance of general nursing care and other supportive measures along with considerations for nutrition and fluid and electrolyte balance cannot be minimised.

In countries with well developed medical facilities, improved type of nursing care and easily available ancillary measures, the modern treatment has ideally centred around the control of spasms and rigidity by neuromuscular blocking agents and maintenance of adequate aeration by controlled respiration in conjunction with tracheostomy. The use of antisera, antibiotics and maintenance of fluid and electrolyte balance, however, are essential.

These methods of treatment are by themselves cumbersome and attended with various complications, and in India they are particularly unsuitable, for the disease is fairly common and the number of skilled anaesthetists is few in any institution. A simple alternative was considered necessary by the author. A combination of drugs which would abolish muscle spasm without affecting respiration or consciousness was regarded as the ideal when combined with certain adjuvant measures. The present study was, therefore, undertaken to evaluate a new method of treatment of tetanus.

METHODS AND MATERIALS

Besides the standard dose of one lac unit of tetanus antitoxic serum and adequate penicillin, sedation with control of spasm was ensured by a combination of chlorpromazine and phenobarbitone. In addition, prednisone or prednisolone and low molecular weight polyvinyl-pyrrolidone were employed. The value of these drugs in tetanus has been reported by Cole and Robertson (1955) ; Kelly and Lawrence (1956) who used chlorpromazine ; and by Lewis *et al* (1954) ; Scheinburg (1952) who employed steroids ; and by Singleton and Witt (1956) who tried low molecular weight polyvinyl pyrrolidone (P.V.P.) with saline for its 'tissue lavage' property. The author has relied on a combination of chlorpromazine and phenobarbitone for sedation and control of rigidity and spasms and has employed steroids and P.V.P. for their added beneficial effects. The care of the wound, if any, considerations of fluid balance and caloric requirements and any need for addition of vitamin supplements were paid due attention to in every case of tetanus and the control group of cases.

The usual adult dose of chlorpromazine was 50 mg. every twelve hours, administered either intravenously, suitably diluted, or intramuscularly varying with the urgency of the case ; and that of phenobarbitone was 3 grains intramuscularly 12

hourly. In some instances a third dose of 50 mg. of chlorpromazine was necessary. The parenteral method of administration was employed for a few days only, and the patient was gradually weaned over to oral medication. In younger subjects the daily dose was calculated on the basis of 1 to 1·5 mg./kg. of expected body weight not weight for weight, and the dose in infants and children was of a higher order. Polyvinyl-pyrrolidone (periston-N) was used by intravenous drip in 100-200 ml. daily doses for a maximum of seven transfusions, and prednisone or prednisolone or its combination has been used in daily dose of 20-30 mg. for a few days only.

The cases were selected from those seeking treatment at Darbhanga Medical College Hospital between September, 1957 and December, 1957 and again between April, 1958 and June, 1958 and covered a total of 91 cases only. Most of the patients were advanced cases showing classical clinical picture and the diagnosis was essentially clinical though in some instances bacteriologic confirmation was also made.

Majority of cases had the standard method of treatment in which various sedatives and muscle relaxants were employed as and when it was thought necessary, in addition to antibiotics and antisera and general nursing care which were common in both the groups. In thirteen cases, in addition to the above, steroids were given, while in twelve cases the author's method of treatment was employed.

The cases with generalised reflex spasm and short period of onset along with shorter incubation period were classed as 'severe', while those in whom there was no generalised reflex spasm at the time of admission were classed as 'mild'. All the patients were treated in the general ward for want of suitable accommodation. The cases in whom the disease was controlled and who had no rigidity even after cessation of therapy were discharged as 'cured', while those who left after admission for some reason or other before completion of treatment, were discharged 'otherwise'. Tables 1, 2 and 3 indicate the severity of the cases, period of stay in the hospital, and the eventual outcome under the particular regime of treatment as compared to other methods of therapy.

Discussion

One is impressed by the type of sedation and control of rigidity attained by the author's therapy. The control of rigidity and reflex spasm with chlorpromazine, particularly when given intravenously, is very striking. This is due to its depressant action on the internuncial neurones in the spinal cord and on the reticular formations in the brain stem (Das Gupta *et al*, 1955). The drug has to be used for a long period in tetanus therapy, but its prolonged use in large doses may be attended with hepatotoxic effects (Robson and Keele, 1956). However, its potentiating effects on other sedatives made it possible to reduce the dosage, as the desired level of sedation was obtained when it was combined with phenobarbitone (Bodman *et al*, 1955 ; Singleton and Witt, 1956). With the recommended dose of chlorpromazine used in the schedule there were little toxic reactions. Intramuscular injections of this drug are painful, and phlebothrombosis expected from its intravenous use has been eliminated by adequate dilution or by its use in a drip infusion. The ease with which such a control can be attained is remarkable and this property has been its most important advantage. In the standard regime of treatment, frequent change from one drug to another is in itself indicative of the ineffectiveness of these drugs, and unless they are pushed to the maximum limit of safety, control of the spasm and the rigidity is wellnigh impossible. Such a bold step is possible only with a team of well trained workers thoroughly conversant with their uses and with all ancillary measures ready at hand.

The adrenal steroids were used by Lewis *et al* (1954), though Scheinberg (1952) had reported its use in tetanus. The cenaesthesia produced with steroids heightens the morale of tetanus patients to undergo successfully through the rigors of the disease, while its antistress activity plays a decisive role in severe cases. The results of treatment are shown in Table 1.

Table 1—Showing Details of Cases Treated under Old Schedule with Added Steroids

Serial No.	Age (years)	Incubation period (days)	Period of onset (days)	Stay in hospital (days)	Result
1	21	9	3	26	Cured
2	21	6	2	3	Died
3	13	4	3	21	Cured
4	35	4	3	21	Cured
5	20	10	5	14	Cured
6	30	9	1	14	Cured
7	18	9	6	20	Cured
8	26	—	4	21	Cured
9	35	7	3	8	Died
10	35	8	2	15	Cured
11	24	5	1	1	Died
12	7	7	3	15	Cured
13	10	15	—	13	Cured

The low molecular weight P.V.P. (periston-N) by virtue of its 'tissue lavage' property was reported by continental workers to be a great advance in all toxi-infectious conditions and at present it is the only means available to us to dislodge the toxins fixed to the tissues. The incorporation of steroids and P.V.P., though expensive, was selected as a means of supplying additional therapeutic advantage in the treatment of tetanus.

TABLE 2—SHOWING COMPARATIVE RESULT OF DIFFERENT REGIMES OF TREATMENT

Treatment schedule	No. of cases	Type of disease			Result		
		Severe	Mild	Cephalic	Cured	Died	Otherwise
Old ...	66	59	7	—	29	30	7
Old with steroid ...	13	9	4	—	10	3	—
Author's schedule ...	12	10	1	1	9	2	1
Total ...	91	78	12	1	48	35	8

TABLE 3—SHOWING DETAILS OF CASES TREATED UNDER THE AUTHOR'S SCHEDULE

Serial No.	Age (years)	Incubation period (days)	Period of onset (days)	Hospital stay (days)	Result
1	10	7	3	16	Cured
2	4	8	2	17	Cured
3	24	5	3	21	Cured
4	28	4	1½	19	Cured
5	35	2	1	19	Cured
6	20	3	4	15	Cured
7	26	7	1	2	Died
8	30	6	2	20	Cured
9	5	7	3	12	Cured
10	40	7	3	12	Cured
11*	45	19	—	1	Otherwise
12	30	3	2	6	Died

* This case was followed throughout the course, even though discharged 'otherwise' from hospital, and had an uneventful recovery.

As will appear from Table 2 the result is very encouraging. The cure rate is over 83 per cent under the author's schedule as compared to just over 43 per cent only under the old method, and about 77 per cent when steroid was added along with the older regime of treatment.

Even the average period of stay in the hospital of patients who survived, was only about sixteen days under the author's regime and eighteen days with the older regime with added steroids as is evidenced in Tables 1 and 3. This if of great importance from the point of national economy with rapidly developing industrialisation programme of our country.

Most of the cases were well-developed and severe, and the effectiveness of the author's method of treatment even in severe cases—even if severity be judged by short 'period of onset' (Brain, 1955)—is quite remarkable. The value of steroids when added to old schedule is quite striking (Table 1). It is noteworthy that most of the deaths occurred in the first few days of hospitalisation and before the patients had the full benefit of hospital treatment. The simplicity, advantage and effectiveness in early trial was so remarkable that this new regime has been accepted for routine treatment of tetanus (Sinha, 1959 ; personal communication).

This regime spares the patient of much of the acute mental anxiety and physical pain and exhaustion due to spasm. At the same time it obviates the necessity of inordinate demand on the nursing and medical staff. This is a great advantage. Further, the total cost of treatment comes to about Rs. 220/- to 250/- only, which is quite reasonable for such a serious infection.

SUMMARY AND CONCLUSION

Only a small number of cases of tetanus has been treated by this regime and comparative result outlined. The efficacy, simplicity and other advantages of the author's regime favour its further trial. Only when tried on a larger number of cases can its true utility be properly evaluated. The use of steroids in tetanus has confirmed its earlier promise.

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TETANUS

S. P. SRIVASTAVA, M.S., F.R.C.S. (ENG.), F.A.C.S. F.I.C.S.

Head of the Department of Surgery

AND

G. G. CHATTERJI, MAJOR, I.A.M.C.

Medical College, Agra

INTRODUCTION

Tetanus is a disease of antiquity. It was described in Edwin Smith Surgical Papyrus supposed to be dated 19th century B.C. but it is an earlier work and dates back to 30th century B.C. (Hussain, 1943).

In 460 B.C. Hippocrates described the prognosis of the disease thus: "Those who are attacked by tetanus either die in four days or if they survive they recover".

Aretaeus, the Cappadocian, described it as "An inhuman calamity, an unseemly sight, a spectacle painful even to behold, not to be recognised by the dearest friends".

The history of the clinical syndrome, the aetiological studies and speculations culminating in discovery of the organism and the final demonstration that human clinical tetanus is almost entirely preventable, is a fascinating story that in many ways parallels the development of modern medicine.

The characteristics of the organism have been described by Humphreys (1935). It has been generally considered that the anaerobic rods with round terminal spores, which are capable of producing characteristic neurotoxin, constitute rather an exceptionally well-defined group. The members only vary slightly from each other. Modern study has made it clear that the limits of *Cl. tetani* are by no means clear-cut as a botanical species. Certain varieties exist, which though highly toxic differ from the accepted types of the species in morphology, motility and biochemical activities. Again, nontoxic variants appear which may or may not agglutinate with each of the known tetanus agglutinating serums, but which morphologically and biologically appear to be identical with the usual forms.

Tulloch (1918) made the original observation that distinct serologic types can be differentiated. This has been confirmed by other workers. Humphreys (1935) described nine agglutinative types. Each is neutralised by the corresponding antitoxin.

In a discussion of clinical tetanus the consideration of the characteristics of tetanus toxin is very important. Ehrlich (1898) separated two distinct and different toxins which are formed independently and are both antigenic.

Tetanospasmin, the more important of the exotoxins, is responsible for the symptoms of tetanus. It is a very powerful toxin. It is carried by parenteral route, is extremely labile and easily destroyed by chemical and physical agents. Toxin production is markedly altered by the symbiotic organisms comprising a mixed culture such as may occur in a wound. Tetanolysin is a haemotoxin, which destroys R.B.C. and leucocytes. Non-pathogenic strains of *Cl. tetani* produce only tetanolysin (Kerrin, 1930; Hall, 1930). While tetanolysin is nonpathogenic it may produce the tetanus infection by virtue of its possible antiphagocytic action which allows the pathogenic tetanus spores and bacilli to grow.

The neurotoxin acts only on nervous tissue with which it unites and by which it can be inactivated *in vitro*. This is known as Takaki-Wassermann reaction (Fullthrope, 1958).

The action of the toxin is also dependent on temperature. If a frog is inoculated with toxin and kept at a temperature below 180°C. tetanus will not develop. If the frog is warmed up it dies of tetanus (Wright, 1955).

The main action of the toxin is on the neural element of the central nervous system. The toxin abolishes or diminishes synaptic inhibition, and is believed to travel first up to the motor nerve trunks and then up the spinal cord (Mayer and Ransom, 1903). This theory of nerve transmission was vigorously opposed by Abel (Wright, 1955) who contended that the toxin was carried to the central nervous system by the blood stream.

More recent work supports the earlier view of Mayer and Ransom (*loc. cit.*). de Antona (1951) showed that an intravenous injection of antitoxin which could protect against a given dose of toxin administered intravenously completely failed to protect against a similar dose of toxin administered intramuscularly. However the same intramuscular dose of toxin failed to kill, if the regional motor neurons to the injected muscle had been divided previously.

Thus it is clear that the toxin injected into the muscle reaches the central nervous system by a route sheltered from circulating antitoxin and in all probability through the motor nerves.

Prophylaxis—An unimmunised patient with suspected wound should be passively immunised (Sherman, 1958). When the age of the wound is 24 hours, 3,000 units of antitetanic serum (A.T.S.) is given and the dose is doubled for each day of

elapsed time up to a total of 10,000 to 12,000 units (Hampton, 1954).

Active immunisation—Brig. A. Sachs, in a Royal Society of Medicine deliberation in October, 1952, suggested introduction of the following method in the British Army. His decision was influenced by observations made about the extremely low incidence of tetanus in the American Army, which supports the value of active immunisation. This is particularly true when the person is being re-inforced with regular booster doses of toxoid and the administration of emergency stimulation dose at the time of receiving the wound. The technique of immunisation is given below :

(a) The primary course consists of 2 injections of tetanus toxoid, 1 ml. each, spaced by not less than 6 weeks or not more than 12 weeks.

(b) The third injection of 1 ml. should be given 6 to 12 months after the second injection.

(c) A booster dose every 5 years should be given, and a booster dose every year if residing in a known tetanus terrain.

(d) A emergency booster dose should be given as soon as the person is injured. The administration of prophylactic tetanus antitoxic serum may still be necessary after receiving the injury in a known tetanus terrain and also in the following circumstances :

(*i*) when there is evidence of no previous active immunisation ;
(*ii*) where there are multiple injuries ; and
(*iii*) where there is considerable delay between infliction of the wound and surgical attention.

Clinical Features

Tetanus is a toxaemia due to infection with *Cl. tetani* as a result of injury which may be trivial. It may be associated with burns, puerperal infection, infection of the umbilical stump and various surgical procedures in which the infection may originate from contaminated suture, dressings and plaster. The incubation period is from 5 to 10 days. The symptoms are characterised by clonic and tonic contractions of voluntary muscles, muscular spasms in the region of local infection followed by trismus which rapidly increases to fixation of jaw ; opisthotonus, and pleurothotonos of muscles may occur. Trismus is said to be due to transmission of the toxin along the facial nerve which is shorter than other spinal nerves and fixation of toxin to the motor endplates.

1. *Chronic tetanus* occurs if prophylactic measures have failed. The incubation period is long (3 to 6 weeks), the symptoms are mild and spasms are few. Trismus is the main symptom.

2. *Local tetanus* is a modified tetanus in which the muscles affected are restricted to one limb, and the spasm affects chiefly the muscles in the region of the wound.

3. *Cephalic tetanus* occurs in two varieties. One is a variety of local tetanus with trismus but without palsy—a result of facial injury, tonsillectomy or otitis media. In the other variety of cephalic tetanus the cranial nerves are affected. This may be due to local or distant infection. There is trismus and palsy of cranial nerves. The mortality in this variety is very high. Ptosis and facial palsy may remain for months. Bulbar tetanus is very rare but is often fatal. It follows visceral infection. There is dysphagia and fatal dyspnoea.

4. *Cryptogenic or idiopathic tetanus* is a mild form of tetanus occurring in the absence of any overt wound. It is said to occur from absorption of toxin from the alimentary tract. Tetanus bacilli have been found in the faeces of 5 per cent of people in urban areas and those dealing with horses.

5. *Puerperal tetanus* is a very severe form of tetanus which occurs from infection during childbirth.

6. *Tetanus neonatorum* is infection of the umbilicus with *Cl. tetani*.

7. *Postoperative tetanus* occurs in epidemic form as a result of inadequate sterilisation of catgut.

8. *Delayed tetanus*—The reopening of old wound, even years after infliction, may stimulate dormant organisms to activity.

Infection through intestinal tract may occur. In some cases the original infection through which the organisms have entered may have healed up before symptoms of tetanus are evident.

Differential Diagnosis

Differential diagnosis should be made for trismus from other causes in the tooth and maxilla and diseases of the temporomandibular joint, from meningitis, strychnine poisoning, tetany, hydrophobia, fibrositis of the back and acute abdomen (if rectus abdominis suffers first).

Materials and Methods

The total number of cases admitted from 1952 to 1958 in S. N. Hospital, Agra, was 97,588. The total number of tetanus cases admitted during the same period was 602. This constitutes 0·61 per cent of total admissions to the Hospital.

TABLE 1—SHOWING CHARACTERISTICS OF EACH CLANICAL GROUP

Items	Mild	Moderately severe	Severe	Fulminating
Trismus	Present	Present	Present	Present
Risus sardonicus	Present	Present	Present	Present
Temperature	Below 98·4°F	Below 99°F	Below 100°F	Above 100°F
Convulsions	Nil	Nil	Present	Present (++)
Localised spasm	Nil	Present	Present (++)	Present (+++)
Incubation period	Over 7 days	Over 7 days	Under 7 days	Under 7 days
Period of onset	Over 48 hours	Over 48 hours	Upto 48 hours	Upto 48 hours
Dehydration	Nil	Nil	Present (+)	Present (++)
Toxaemia	Nil	Nil	Present (+)	Present (++)
Complication	Nil	Nil	Present (+)	Present (+)
Summed up clinical evaluation ...	Satisfactory	Fair	Poor	Very poor

All the cases admitted after April, 1959 (100 cases) were subjected to a thorough history taking and allotted a group which was arrived at by prognostic criteria. The study also included 602 cases seen during the period 1952-58.

The cases were grouped according to the severity of the signs and symptoms as mild, moderately severe, severe and fulminant. The characteristics of the clinical groups are shown in Table 1 from which it may be noted that all neonatal cases were put under the fulminating group ; all patients over the age of 40 were put under the severe group ; and all cases in whom the summation of incubation period and period of onset were less than 7 days, and whose prognosis presented a grave or poor outlook were put under the fulminating group.

TREATMENT

The treatment adopted for sedation and control of convulsions is considered under the following groups :

GROUP 1 :

In adults, the following drugs in the order of priority are used : chlorpromazine in doses of 50 mg., I.M., promethazine in doses of 50 mg., I.M. and phenobarbitone (gardenal) sodium in doses of 1 to 3 grains, I.M.—all 6 to 8-hourly.

In infants, chlorpromazine is given in doses of 6 mg. per lb. body weight in 24 hours, e.g., for 8 lb. infants 48 mg. in 24 hours. Phenobarbitone sodium, 1 gr., I.M. stat, and later 1 gr., 8-hourly.

A paediatrician should be consulted for treatment of lung or abdominal complications, e.g., bronchopneumonia or diarrhoea.

GROUP 2 :

Paraldehyde—This is to be given preferably by deep intramuscular injection in 4-5 c.c. doses from an ampoule which is available in the market. Rectal administration of paraldehyde is not safe, it causes local irritation. Paraldehyde is inferior to the drugs mentioned above.

GROUP 3 :

For pain and restlessness, administration of pethidine, 50 to 100 mg I.M. or I.V., is very helpful in keeping the patient quiet. It is also very useful when phenobarbitone cannot be given by mouth. Phenergan or luminal can also be given intramuscularly.

All intramuscular injections should be given deep in the gluteal region. Intravenous injection of largactil 50 mg. with pethidine 50 mg. is very useful in controlling spasm. The former can also be added to the intravenous saline drip.

GROUP 4 :

Antitetanic serum (A.T.S.)—Since the last war the practice has been to give one lac units of A.T.S. intravenously. This maintains sufficient concentration of the serum in blood to neutralise the toxins for one week. The intravenous injection of one lac units has to be repeated if the symptoms still persisted.

It has been said that the toxin after uniting with the neural tissues changes its character to a type which cannot be acted upon by the antitoxin in A.T.S. Only the toxin which has not been fixed in neural tissues could be neutralised by the antitoxic serum. Hence the futility of administering A.T.S. in all types of cases.

An antibiotic, such as penicillin, should also be given in large doses, as it acts both on mixed infections as well as on *Cl. tetani* which grows in symbiosis with other organisms.

Forbes and Auld (1955) treated 22 cases without giving A.T.S. without any mortality. Similar is the experience of other surgeons. In moder-

ately severe or severe and fulminating cases with infected wound, one lac units of A.T.S. should be given by intravenous drip in a pint of saline.

As a routine, it is difficult to withhold completely the administration of A.T.S. as the figures of mortality are found to vary ; but the doses of the antiserum can be reduced because too big a dose is unnecessary when the toxins have been fixed in the nerve tissue. The dangers of anaphylaxis and serum reactions should also be borne in mind.

Before giving an injection, the patient should be tested for serum sensitivity. This is done by intradermal injection of 1/10 ml. of serum in 1 : 10 dilution. If there is no reaction in half an hour, 40 thousand units of serum is injected intramuscularly, and the rest (60,000 units) is diluted in 450 ml. of normal saline and injected in slow intravenous drips. The serum administration should take about two hours.

A.T.S. is not generally given to 'mild' group patients. Serum is given to all other groups in varying doses. The doses vary from 20 thousand to one hundred thousand units. In mild cases the administration of A.T.S. could be omitted completely and the progress watched. In severe and fulminating cases without wounds or healed scars, the dose of serum could be cut down to half, i.e., 50,000 units only.

GROUP 5 :

Further therapy is aimed at reducing the reflex spasm. Mephenesin relaxant, 5 to 10 ml. of a 10 per cent solution, is given intramuscularly. Severe convulsions are controlled by intravenous drip administration of 4 g. of mephenesin in 500 ml. of glucose-saline. The drip rate is adjusted to the need of the patient. This requires constant watch.

Apnoeic crisis—This is controlled by injecting, 2 per cent mephenesin I.V. through the drip tubing. A 10 ml. syringe loaded with 2 per cent mephenesin solution must be kept ready by the patient's bedside for emergency use.

GROUP 6 :

Antibiotics like penicillin, streptomycin or other broad spectrum antibiotics should be given to combat secondary wound infection, when present.

The treatment of the wound is carried out by dressing with antiseptic lotion, after preliminary washing with hydrogen peroxide. Radical excision of the wound is not necessary, except for debridement and for cleaning dead tissues of dirt and foreign matter.

GROUP 7 :

Nursing care is very necessary. Evacuation of bowels by enema should be attempted after giving necessary sedatives. Nasal feeding may also be required. The patient should be kept in a dark room without any disturbance or noise.

GROUP 8 :

Prophylactic tracheostomy is advocated in severe or fulminating cases to prevent asphyxia due to laryngeal spasm. In the authors' series the patients were not willing to submit to operation, and hence the benefits of this procedure could not be tried. Diaphragmatic spasm is relieved by muscle relaxants.

GROUP 9 :

The latest method of controlling the spasm is the complete paralysis of the skeletal system by muscle relaxants. Controlled respiration could be carried out for days. Parenteral administration of fluids and dextrose with vitamins, is necessary to maintain the nourishment. Electrolyte study of blood serum should be done frequently, and breathing should be maintained by automatic respirator under proper medical supervision.

OBSERVATIONS

The data obtained from the observations made on the cases of tetanus under review [1952-1958 series and 1959 (April to October) series] are analysed in relation to different factors and presented in tabular form (Tables 2-17).

TABLE 2—SHOWING ANALYSIS OF THE CASES IN RELATION TO SEX

Tetanus cases	1952-58		1959	
	Total cases	Percentage	Total cases	Percentage
Male ...	403	66·95	61	61
Female ...	199	33·05	39	39
Total ...	602	100	100	100

TABLE 3—SHOWING ANALYSIS OF THE CASES IN RELATION TO AGE

Age group (years)	1952-58		1959	
	No. of cases	Percentage	No. of cases	Percentage
Above 12 ...	283	47	58	58
Up to 12 ...	319	53	29	29
Neonatal ...	—	—	13	13

TABLE 4—SHOWING DISTRIBUTION OF THE CASES ACCORDING TO LOCALITY

Place of residence	1952-58		1959	
	No. of cases	Percentage	No. of cases	Percentage
Rural	480	80	60	60
Urban	122	20	40	40
Total ...	602	100	100	100

TABLE 5—SHOWING ANALYSIS OF THE CASES IN RELATION TO THE TYPE OF WOUND

Type of wound	1952-58		1959	
	No. of cases	Percentage	No. of cases	Percentage
Contused wounds ...	260	45	14	14
Punctured wounds ...	42	7	20	20
Postpartum and post-abortal ...	49	8·11	12	12
Otorhoea ...	59	9·5	11	11
Burns ...	9	1·51	10	10
Umbilical sepsis ...	97	16·1	13	13
Boils, cancer, etc. ...	34	5·71	10	10
Undetermined ...	52	8·4	10	10
Total ...	602		100	

TABLE 6—SHOWING ANALYSIS OF THE CASES IN RELATION TO THE SITE OF INFECTION

Site of infection	1959	
	No. of cases	Percentage
Lower limb	16	16
Upper limb	15	15
Uterus	12	12
Chronic suppurative otitis media...	11	11
Head and neck	4	4
Male genitalia	1	1
Female genitalia (cancer cervix) ...	2	2
Umbilical sepsis	13	13
Trunk	6	6
Undetermined	20	20
Total ...	100	100

TABLE 7—SHOWING ANALYSIS OF THE CAESS IN RELATION TO THE DURATION OF ILLNESS

Duration of illness (days)	1952-58		1959	
	No. of cases	Percentage	No. of cases	Percentage
Less than 4 ...	49	8	19	19
4 to 10 ...	312	50	34	34
More than 10 ...	181	30	17	17
Undetermined ...	60	10	30	30
Total ...	602	100	100	100

TABLE 8—SHOWING ANALYSIS OF THE 1959 GROUP OF CASES IN RELATION TO INCUBATION PERIOD

Incubation period	No. of cases	Percentage
0 to 48 hours	62	62
More than 48 hours	17	17
Without generalised spasm and convulsions	21	21
Total ...	100	100

TABLE 9—SHOWING MORTALITY RATE IN RELATION TO THE DOSAGE OF A.T.S.

Doses of A.T.S. (units)	No. of cases	No. of deaths	Mortality per cent
0 ...	8	0	0
20,000 ...	15	3	20
30,000 ...	1	0	0
40,000 ...	12	3	25
50,000 ...	7	2	28·5
60,000 ...	6	0	0
70,000 ...	Nil	0	0
80,000 ...	1	0	0
90,000 ...	Nil	0	0
1,00,000 ...	47	24	51·6
1,20,000 (Outside cases)	2	1	50

TABLE 10—SHOWING THE ANALYSIS OF THE RESULTS OF TREATMENT

Result	1952-58 cases		1959 cases	
	No.	Per cent	No.	Per cent
Cured ...	293	48·7	60	60
Expired ...	295	49	34	34
Left against medical advice ...	14	2·3	6	6
Total ...	602	100	100	100

TABLE 11—SHOWING RELATION OF CURE RATE WITH INCUBATION PERIOD IN 1952-58 AND 1959 SERIES

Incubation period	No. of cases	No. surviving	Cure rate (percentage)
1952-58 SERIES			
9 days or less ...	306	97	32·35
10 days and over ...	236	170	71·65
Undetermined ... (No history of injury)	60	40	66·66
1959 (APRIL-OCTOBER) SERIES			
9 days or less ...	47	27	57·44
10 days and over ...	34	28	82·35
Undetermined ...	19	12	63·18

TABLE 12—SHOWING RELATION OF TRISMUS AND SPASM WITH MORTALITY

Interval between trismus and spasm (generalised)	No. of cases	No. of deaths	Mortality per cent
0-48 hours ...	62	21	33·67
Over 48 hours ...	17	4	23·53
Cases without any generalised spasm	21	5	23·80

TABLE 13—SHOWING MORTALITY ACCORDING TO SEX (NEONATAL SERIES)

Neonatal series	No. of cases		Mortality per cent
	Male	Female	
1952-58 :			
No. of cases treated	72	22	94
Cured ...	14	4	18
Deaths ...	58	18	76
Mortality (%) ...	77·1	80	77·7
1959 :			
No. of cases treated	11	2	13
Cured ...	9	0	9
Deaths ...	2	1	3
Left against medical advice ...	—	1	1
Mortality (%) ...	18·1	50	30·10

TABLE 14—SHOWING MORTALITY RATE IN POSTPARTUM AND POSTABORTAL TETANUS (PEURPERAL CASES)

Period	1952-58	1959
Total No. of cases	602	100
No. of cases of postpartum and postabortal tetanus	49	12
Cured	13	4
Deaths	36	8
Mortality (%)	73·46	66·66

TABLE 15—SHOWING RESULT OF TREATMENT WITH ANTISERUM

Antiserum	Lived	Died	Total	Mortality per cent	
					Jhonstone's series
Given ...	58	33	92	35·8	33
Not given ...	8	0	8	0	63

COMMENTS

Prophylaxis of all children with triple antigen vaccine at home or in school is essential.

Injection of toxoids for mass inoculation in rural areas and in factories and schools, or passive immunisation by A.T.S. (3,000 units) after the slightest injury, in primary health centres is very necessary in villages. It will save the cost of treatment by A.T.S. and reduce the admission of tetanus cases (severe and mild) to hospitals.

There should be provision of special tetanus wards in hospitals with trained nursing staff, with complete isolation in dark and silent cubicles or rooms.

TABLE 16—SHOWING RESULT OF VARIOUS TYPES OF TREATMENT IN 100 TETANUS CASES

Type of treatment	Left against medical advice	Cured	Died	Total	Mortality per cent
A.T.S.+antibiotic+largactil+phenergan+phenobarbitone	2	14	5	21	23·8
A.T.S.+antibiotic+largactil+phenergan+paraldehyde	—	3	1	4	25·0
A.T.S.+antibiotic+largactil+phenergan+myanesin	—	7	5	12	41·6
A.T.S.+antibiotic+largactil+phenergan+phenobarbitone+myanesin ...	—	9	8	17	47·1
A.T.S.+antibiotic+largactil+phenergan+pethidine	1	3	3	7	42·8
A.T.S.+antibiotic+largactil+phenobarbitone	3	6	2	11	18·1
Antibiotic+largactil+phenergan+phenobarbitone	—	5	—	5	0·0
Antibiotic+largactil+phenergan+paraldehyde	—	1	—	1	0·0
Antibiotic+largactil+phenergan+myanesin	—	3	—	3	0·0
A.T.S.+antibiotic+phenobarbitone	—	1	—	1	0·0
A.T.S.+antibiotic+paraldehyde	—	4	4	8	50·0
A.T.S.+antibiotic+paraldehyde+myanesin	—	4	6	10	60·0
Total	6	60	34	100	

TABLE 17—SHOWING CAUSES OF DEATH

Causes of death	No. of cases	Mortality per cent
Asphyxia	11	33·33
Hyperpyrexia	4	12·15
Pulmonary infection ...	2	6·06
Sensitivity reaction to serum ...	8	Nil
Toxaemia	9	24·24
Respiratory failure ...	7	21·21
Cardiac arrest	1	3·03
Total ...	34	100·00

It is not necessary to administer A.T.S. to mild cases and cases of local tetanus. Regarding fulminating types and severe types, A.T.S. may be given in doses of 1 lac units only to neutralise the circulating toxins in the blood. Serum therapy has no effect on the toxins fixed in the neural and spinal cord tissues.

Largactil and phenergan, with or without phenobarbitone, have improved the cure rate, with avoidance of complications resulting from paraldehyde and magnesium sulphate (25 per cent) administration by intramuscular injections.

Myanesin (mephenesin) has been found very useful in controlling tonic rigidity and frequent convulsions which may give rise to laryngeal spasm or rupture of muscles.

Mortality rate has improved in tetanus neonatorum by administration of largactil and phenobarbitone along with injection of A.T.S. 20,000 units I.M., where umbilical sepsis is present.

Heavy doses of antibiotics with cleaning of the wound with hydrogen peroxide are essential. Excision of wound is not necessary except for cases of gangrene and for debridement.

In fulminating cases tracheostomy has a beneficial effect as a prophylactic against apnoeic crises.

Mortality in cases of tetanus depends on the incubation period and the period of onset. The prognosis is grave if the incubation period is less than 7 days and period of onset less than 48 hours.

Treatment by curare and ganglion-blocking drugs along with controlled respiration by intratracheal intubation or tracheostomy is worth a trial in the fulminating group of cases.

SUMMARY

A review is given of the literature on tetanus including immunisation.

The clinical features in different clinical types of tetanus, their differential diagnosis and the methods of treatment are given.

Data obtained from the study of 606 cases during the period between 1952 and 1958 and of 100 cases in 1959 are analysed in relation to different factors.

The results of treatment with A.T.S. and other drugs are recorded.

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PRACTITIONERS' SERIES

VOMITING IN INFANCY AND CHILDHOOD

O. N. BHAKOO, M.B., B.S., D.C.H.

AND

PRAN N. TANEJA, M.D., M.R.C.P., D.C.H.

Department of Paediatrics
All-India Institute of Medical Sciences, New Delhi

Vomiting or ejection of the gastric contents through the mouth is a common complaint in the paediatric age group. A clear understanding of its genesis and the aetiological factors causing it should be well understood by every practitioner who has the responsibility of looking after children.

The act of vomiting begins with closure of the pylorus and constriction of the antrum with concomitant relaxation of the stomach, cardia and oesophagus. Respiration is inhibited, the glottis closes, and the soft palate is elevated to shut off the nasopharynx. Now forceful contraction of the diaphragm and abdominal muscles causes vomiting. Because of poor co-ordination in infants and young children, the vomitus often comes through the nose as well as the mouth. Hence there is a real danger of aspiration, especially so in premature infants and in those who have neuromuscular disability.

But vomiting is not so forceful in all cases, especially in small infants who with passive act and without much force not uncommonly bring out a little milk into their mouths immediately after feed. This is known as 'regurgitation' or 'posseting' or 'spitting up'. It occurs in all babies. It is generally harmless though in some babies it happens so frequently that there may be substantial loss of nutrition. Excessive regurgitation characteristically occurs in a highly active baby who exhibits unusually rapid movements of the arms and legs, and is less often seen in a placid baby. It is commoner in the first 2 to 3 months than later but it may become troublesome in the second six months of the baby's life.

It is very common for milk to be expelled with considerable force, when wind is also brought up. Any cause, therefore, of excessive swallowing of air will lead to this condition. It is a matter of opinion whether this should be called posseting or vomiting. It does not matter what it is called, as long as its nature is understood. Posseting causes much worry to the mother who often exaggerates in stating the quantity of milk brought out by the child. The doctor should, therefore, observe the state of hydration of the child and make a study of the child's weight in relation to his age and weight at birth. Usually one finds that in spite of the mother's complaint that there is severe and frequent vomiting, the child is in no way dehydrated or below the expected weight. A few causes of regurgitation are enumerated:

1. *In breast-fed babies*—Retracted nipples, scanty supply of milk or prolonged sucking after the milk has been obtained resulting in excessive swallowing of air and in the wake of its expulsion the milk is also ejected.

2. *In bottle-fed babies*—Too small or too large a hole in the nipple or partial filling of the nipple during feeding or else continued sucking when milk in the bottle has been finished.

3. Improper posture after feeding, e.g., when the baby is made to lie on the left side, or excessive handling after the feed.

4. Cod-liver oil may make a child sick and he may spit it out.

5. Use of 'pacifiers' (rubber nipples which are given to the babies for sucking so that they keep quiet) leads to a lot of air swallowing.

At times chalasia or cardio-oesophageal relaxation as a result of incompetence of the gastro-oesophageal valve mechanism leads to regurgitation. It usually starts on the 3rd to the 10th day of life. Regurgitation usually does not occur until the infant is put down and often it continues for weeks or a few months. Ultimately the symptom gradually disappears.

Hunger is at times a cause of posseting. An infant who is not satisfied is likely to be restless after feeding. He is apt to suck other objects within reach and thereby swallow considerable air which leads to vomiting. The symptoms are often mistaken for those of indigestion and the food is reduced; whereas if it is increased and enough food is given to satisfy the child, he will remain quiet and may soon fall asleep.

Vomiting in Neonatal Period

In the first few hours after birth newborns at times vomit mucus, often blood-streaked. This vomiting frequently persists after the first few feeds. It may be due to irritation of the gastric mucosa by the material swallowed during delivery. If vomiting is protracted, gastric lavage with physiological saline solution may relieve it.

Most of the vomiting occurring in the neonatal period is due to regurgitation. When vomiting occurs shortly after birth and is persistent the possibility of increased intracranial tension or of intestinal obstruction must be considered. The different causes of vomiting are:

1. *Oesophageal obstruction*—In complete oesophageal obstruction there is an unusual drooling from the mouth and vomiting occurs with the first feeding. A feeling of resistance in passing the catheter into the stomach clinches the diagnosis.

2. *Cardiospasm*—There is a functional spasm of the cardiac sphincter and radiological demonstration of the obstruction at the cardiac end of the oesophagus without organic stenosis is diagnostic. Chalasia has already been discussed.

3. *Intestinal obstruction*—The age at which the vomiting starts is related to the site and degree of obstruction. In small gut obstruction vomiting occurs within a few hours, while in large bowel obstruction as in imperforate anus, vomiting may start as late as 24 to 48 hours after birth. Vomiting is frequent, persistent, non-projectile, copious and unless the obstruction is above the ampulla of Vater, bile-stained. An associated abdominal distention, visible deep peristaltic waves over the abdomen and constipation are present. X-ray film in upright position aids in finding the site of obstruction by noting the distribution of air. Normally air reaches the large gut within 3 hours after birth. The absence of lanugo or cornified epithelium in the meconium further strengthens the diagnosis of complete intestinal obstruction. A rectal examination must be made in all suspected cases of intestinal obstruction.

4. *Congenital diaphragmatic hernia*—This is a very rare condition in which abdominal contents (often stomach, small intestine and sometimes spleen) are found lying in the thorax, with the result that the abdomen looks rather flat, and the mediastinum becomes shifted and the baby becomes dyspnoeic and cyanosed immediately after feeding. The thorax is tympanitic on percussion on that side and intestinal sounds may be heard on auscultation.

5. *Pyloric stenosis*—Vomiting may begin any time after birth, but usually it does not assume its characteristic projectile pattern before the second or third week. Perhaps in no other condition is there such forceful vomiting projecting forwards by 2 to 4 feet. Vomiting occurs during or shortly after a feed. It may occur after every feed or after 2 to 3 feeds. There is apparently no nausea, and the infant will take another feed immediately. The vomitus consists only of acidic curdled gastric contents which may be tinged with blood. It is never bile-stained. The associated gastric peristaltic waves from left to right and palpation of the pyloric tumour below the costal margin, which can be palpated best just after vomiting when the infant is being fed, help in the diagnosis.

6. Anorexia, constipation, abdominal distention and vomiting in the absence of a readily demonstrable organic obstruction are suggestive of congenital megacolon in the newborn infant.

7. *Intracranial birth injuries*—Here associated history of prolonged or obstructed labour, presence of rigidity and hyperreflexia, tense fontanelle and absent Moro's reflex* help a diagnosis.

8. *Infections*—Especially septicaemia, meningitis and urinary tract infections should be looked for.

Vomiting Beyond Newborn Period

Vomiting may be associated with diarrhoea as in acute gastroenteritis. It is a serious condition, because additional loss of water and electrolytes is further hazarded by inability for oral feeding, with the result that severe dehydration and electrolyte imbalance soon supervene.

Not uncommonly a male child 7 to 8 months of age may be brought with complaints of vomiting of sudden onset with severe paroxysmal pain and restlessness followed, within a few hours, by passing of blood and mucus per rectum. On abdominal palpation, an elongated sausage-shaped tumour may be felt in the distribution of the colon. These cases of 'intussusception' should not be confused with ordinary gastroenteritis. On the other hand, atypical cases of intussusception are not uncommon. When the obstruction is intermittent and incomplete, blood-stained discharge per rectum may not be found, the presenting symptom being intermittent abdominal pain. Diagnosis in these cases depends upon radiological examination after a barium enema.

In acute peritonitis vomiting is an early feature, but it may continue even in late stages due to paralytic ileus. The same is true for appendicitis.

Vomiting in this age period may be due to the following conditions:

Infections—In any acute infection vomiting may be an important initial symptom. The commonly overlooked infections are those of the throat, middle ear, and urinary tract. Toxic vomiting may occur in uraemia and diabetic acidosis.

Organic nervous diseases—Vomiting is an early feature of meningitis and encephalitis. In space-occupying lesions (intracranial neoplasm, cerebral abscess, intracranial haemorrhage or subdural hematoma) vomiting is persistent, projectile, aggravated by a change in the posture, e.g., more so on rising in the morning and is not associated with nausea. Rarely it may be non-projectile and associated with nausea.

Reflex vomiting—Conditions simulating 'gag reflex' may lead to vomiting, e.g., copious postnasal drip, enlargement of the uvula, persistent or spasmodic cough (pertussis). Gagging after food may occur in infants started too early on solid foods or where solid foods are too long delayed.

Vomiting occurring before breakfast, associated with nausea and a sense of fainting may be due to hypoglycaemia.

Nervous vomiting—It is seen in car sickness or sea sickness and that following swinging. It is said to be more common in neurotic children but the role of visual and aural disturbances cannot be ignored.

Intestinal obstruction—The features of intestinal obstruction have already been discussed.

Recurrent vomiting—It is also called 'cyclic', 'periodic' or 'acetonaemic' vomiting. It starts usually between 2 to 4 years of age, but can occur any time from six months to ten years and often subsides as the child enters adolescence. It recurs at intervals ranging from 1-2 months to 2-3 attacks a year. It is common in intelligent children, the trigger mechanism being fatigue, emotional excitement, onset of an acute infection and fasting from any cause. The onset of an attack may be sudden with vomiting from the

* Moro's reflex is a symmetrical abduction of the upper limbs when there is a sudden noise or the child is suddenly moved by a bang on the bed or the cot. Normally it disappears by 3 to 4 months of age.

beginning, or there may be a prodromal period of hours or a day during which there is malaise, irritability, anorexia, and constipation.

Vomiting is often forceful, with much retching, and occurs whenever anything is swallowed and even when no food has been taken. As the attack continues, prostration becomes marked and ketosis may result due to prolonged starvation. There may be history of migraine in the family or migraine may subsequently develop in the patient in later life. A neurogenic contributory cause should always be kept in mind.

Before labelling this diagnosis other causes of recurrent vomiting in childhood must be excluded —the common ones being recurrent appendicitis, subacute recurrent intestinal obstruction (intestinal tuberculosis, roundworm infestation, etc.), brain tumours, recurrent urinary infections (usually based on congenital malformations) and rarely food allergy.

Psychogenic Vomiting

This is met with at all ages, even in young infants. It may be a symptom of mild emotional maladjustment or one manifestation of a serious neurotic disorder. It is brought about by a variety of circumstances.

Excessive crying may terminate in vomiting. Children may vomit unpalatable food or the food with unpleasant associations or else if they are forced to eat. It may be an 'attention-seeking device' or a response to tense and hostile parents. If vomiting is the subject of unwise conversation in front of the child, and if he realises that there is a great deal of anxiety about it, it is likely to recur, partly as an attention-seeking device and partly as a result of suggestion.

At times a child may get vomiting on a 'school morning' due to dread of going to school which may be related to school work or an unpopular teacher. The vomiting pattern as an expression of personality difficulties often goes back to early gastro-intestinal upsets, to regurgitation from air swallowing or forcing of food by an overzealous mother.

Regardless of the presence of psychogenic factors, a thorough physical examination must be made to rule out other causes of vomiting. The discovery of physical disease, on the other hand, does not settle the causal relationship. Most difficult to evaluate are cases in which both somatic and psychic factors operate.

Rumination

Rumination, also called merycism, is a habit acquired by babies usually after the age of three months. Child is brought to the doctor on account of vomiting. The diagnosis can sometimes be made on history alone. The child retches, puts his finger into the mouth and brings up the milk in the oral cavity. Some of the milk dribbles out, while the rest is swallowed. The child enjoys it. Its cause is not understood but in most instances it is due to a disturbed parent-infant relationship, particularly to a lack of affectionate care.

Diagnosis

In a case of vomiting answers to the following questionnaire may help in the diagnosis.

1. Whether quantity of the vomitus is appreciable.
2. Whether the material is regurgitated in small amount or brought up all at once.
3. Whether vomiting is forcible and projectile.
4. Character of vomitus—bile-stained, bloody or faecal.
5. Relation to meals.
6. Presence of nausea as determined by appetite.
7. Presence of gastric peristalsis.
8. Any other evidence of disease, e.g., pain, fever, prostration. Observing the child during the act of vomiting may help.

Treatment

Regurgitation—Excess of swallowed air can be got rid of by what is called 'breaking of wind', i.e. the baby is held upright over the shoulder for a few moments after the feeding and patted gently on the back until belching occurs. Occasionally, it is necessary to interrupt the feeding for this purpose. After the feed the baby should be allowed to lie on the right side for sometime, with the head slightly raised. The baby should not be played with during this period, but should be propped up during feeding so that the air collects near the cardiac end of stomach and can be easily expelled. One should avoid feeding the baby in the horizontal position. The hole in the nipple should be adequate to let out 20 to 30 drops every minute. The routine use of 'pacifiers' should be discouraged.

In chalasia the vomiting can be prevented almost entirely if the baby is held in upright position for about 30 minutes after feeding.

In obstructive lesions of the intestine surgical help should be sought.

Rumination—Various devices used for this condition are:

1. Distract child's attention after each feed.
2. Make the feeds thick.

3. Use a tight fitting cap with a strap which passes under the jaw and prevents the opening of the mouth.

4. The mother should be tutored about the need of the baby for tender love and care.

Symptomatic treatment may be tried where the aetiological factor cannot be determined. Useful drugs are chlorpromazine (largactil) or one of the phenothiazines (phenergan). Mild sedation may sometimes be helpful.

In less severe cases of pyloric stenosis especially where a pyloric tumour cannot be felt, use of atropine derivatives or 'antrenyl', sometimes brings about relief.

Last but not the least, the role of parenteral fluid and electrolyte therapy should not be underestimated especially in cases of prolonged and persistent vomiting where the fluid and electrolyte loss may be sufficient to produce dehydration and metabolic alkalosis.

SPECIAL ARTICLE

LATHYRISM : THE OLD AND NEW CONCEPTS

D. N. SHARMA, M.B.B.S. (PAT.), PH.D. (ST. ANDREWS)

Department of Physiology
Darbhanga Medical College, Laheriasarai

Lathyrism is a syndrome caused by the ingestion of grains of the lathyrus species. This definition is as simple as misleading, as we shall see presently. If lathyrism is to be defined as a condition caused by eating lathyrus, one can easily argue that it is in fact lathyrism whether it is caused by *L. sativus* (*khesari*) in man or by *L. odoratus* (sweet pea) in rats. But unfortunately things are not so simple. Vivanco and Jimenez-Diaz (1951) preferred to use the term 'odoratism' for the condition caused by administration of *L. odoratus* in rats, and to reserve the term 'lathyrism', for the condition caused by *L. sativus* in the human. For some time this definition seemed to settle the issue. But difficulty again arose. It is now well known that the typical lesions due to *L. odoratus* feeding of rats can also be produced by administering to them (rats) many synthetic nitriles. The characteristic lesions of sweet pea feeding can also be duplicated by feeding several other pulses, e.g., *L. pusillus*, *L. hirsutus* and *L. tingitanus*. Selye (1957), therefore, suggested a working method by calling human lathyrism as neurolathyrism and rat lathyrism as osteolathyrism. The following are brief accounts of the two types of lathyrism to facilitate their comparison :

Neurolathyrism occurs in man and sometimes in horses. In man, the signs and symptoms in an established case of lathyrism are spastic paralysis of the legs, exaggerated knee jerks, exaggerated ankle jerks, sometimes ankle clonus and scissors or waddling gait—all pointing to a lesion of the upper motor neurone. The disease is of an unknown pathogenesis but it is an affection essentially of the nervous system. In horses, neurolathyrism manifests itself by affecting their hind limbs. The horses become lame, 'gone in the loins', have dyspnoea and start 'roaring'.

In spite of the claim by various investigators, neurolathyrism has not been conclusively produced in the laboratory and we do not know the active principle in the pulse that causes it. On the other hand, osteolathyrism can be produced at will in rats by feeding *L. orodatus* or *L. pusillus*. The active principle has been isolated as β-aminopropionitrile. The disease can also be reproduced by administration of various other nitriles and non-nitrile compounds. When the disease develops in animals, they lose weight, become lame, their skin becomes thin, their fur loses lustre and becomes unkempt, sparse and rumpled. In short, the disease affects mainly the mesenchymal tissues. It is mostly a disease of the skeleton. Curiously enough *L. sativus*, *L. cicera* and *L. clymenum*, which have been alleged to cause epidemics of lathyrism in man, are healthy food for rats who thrive well on them.

A brief historical background of the two varieties of lathyrism is given below.

Human lathyrism—Ever since Hippocrates wrote : "At Ainos all those, both men and women, who ate pulse (legumes) regularly lost control of their lower limbs and this condition persisted," people began to look for the presence of the disease in various countries. Chopra (1938) pointed out that it was mentioned in *Bhavaprakash*, which was written in 1550. In the *Madhava-Nidan* (The Aetiology and Pathology of Hindu Medical Science) occurs a couplet which, when translated, means that when the patient attempts to walk he trembles, there is loss of co-ordination, the disease is called *'Kalaya-Khanja'* , lameness from pulses. Amongst the earliest observers, the names of Ramazinni (1691), Chevallier (1841) and Sleeman (1844) are worth mentioning. Strange enough, Sleeman (1844) was a non-medical man who observed a large number of cases in the then Central Provinces (now Madhya Pradesh) of India. Vilmorin (1847) reported the incidence of the disease in Bourgueil (Indre-et-Lorie) in France.

Irving (1859) published a series of papers on cases seen in what is now Uttar Pradesh in India. Cantani (1874), for the first time gave the name lathyrism to the disease, and reported that the disease was known under various local names, viz., Crurum exsolutio, Crurum impotentia, Crura imbecillia and Storpoidelle gambe. Mericourt (1883) confused lathyrism with beri-beri and it was Proust (1883) who tried to remove the confusion. The German worker Schuchardt (1885-87) thought that certain legumes which were quite innocuous became harmful after being damaged. Bouley (1883) observed the presence of the disease in horses, while Gabory (1883) found that when ducks and peacocks were fed with *L. cicera*, they became sleepy and died very quickly.

While investigators on the continent were thus discussing the disease, a curious incident took place in Liverpool (Leather and Sons, 1885). Some horses belonging to a team owner, became suddenly ill and suffered from a peculiar and apparently epidemic disease. The diseased horses appeared to be in perfect health on rest but on moving them in the stall, they staggered somewhat and had imperfect control over their legs. The horses when taken out of the stall and exercised, were at once seized with dyspnoea and roaring and in many cases died instantly due to asphyxia. When the cause was investigated, it was found to be due to consumption of Indian *mutters* (*L. sativus*) by the horses. The equine lathyrism was also observed by McCall (1886). A sensational case was tried at the Bristol Summer Assize, before Mr. Justice Lawrence between the Bristol Tramways and Carriage Company *vs.* Weston and Company (Anonymous, 1894). The case arose out of the fact that Weston and Company had sold Indian peas (*L. sativus*) to the plaintiff and as a result of feeding the horses with the peas, no fewer than 127 were attacked with illness and some of them died. The main symptoms were staring of the eyes and apparent strangulation ; the horses would fall down and would be panting for breath. They also showed a general disturbance of the digestive system, exhaustion, emaciation and roaring.

An excellent account of human lathyrism in Russia was given by Spirtov (1903), who reported that earlier epidemics were also observed by Shabalin, Semidelov and Muchina. Spirtov (1903) claimed to have produced the disease in dogs by feeding them on 3/4 part of *L. sylvestris* in their diet. According to Spirtov (1903) Kojewnikoff, who wrote a book in 1894 on lathyrism, stated that cold and vetch caused the disease. Buchanan (1904) published a very comprehensive report after collecting various data about the disease, as found in India. In 1896-97, during a terrible famine in India, Hendley (1903) could easily collect one to two hundred such cases in a famine relief camp of 4,000 persons. In a letter to Buchanan, he wrote : "I have seen 190 cases this morning". Stockman (1917 a, b) made a detailed study of lathyrism as a whole and lathyrism as found in man. Buzzard and Greenfield (1921) performed postmortem examination of a lathyritic patient and found that the lesions were very similar to those occurring in ergotism. In the lumbar and thoracic regions, the margins of the spinal cord showed a 'honeycombed structure' as is seen in subacute combined degeneration. Acton (1922) and Acton and Chopra (1922) conducted researches into the pharmacological action of *khesari* amine and into the causation of lathyrism in man. Vazeux (1923) observed that feeding of cattle with *L. clymenum* grains produced an epidemic of lathyrism. Visco (1923, 1924 a, b) found that none of the rats fed with lathyrus grains, namely *L. sativus*, *L. cicera* or *L. clymenum* developed lathyrism. Zagami (1932) confirmed the findings of Visco (1924b) when he found that white rats fed on *L. sativus*, though showing slow growth, did not develop lathyrism. Anderson *et al* (1925) carried out a chemical examination of the seeds of *L. sativus* and found that they did not contain any alkaloidal substance. Controlled experiments with this over long periods in ducks and monkeys demonstrated that the grains were harmless and provided a nourishing diet for them. The examination of akta (*Vicia sativa, L. var. angustifolia*) revealed that it contained bases showing alkaloidal properties. Anderson *et al* (1925) isolated two such bases, vicine and divicine and a cyanogenetic glucoside vicianin. McCarrison (1926) noted that the disease was confined entirely to males. McCarrison (1928), McCarrison and Krishnan (1934) further confirmed that *L. sativus* did not cause lathyrism in rats. Megaw and Gupta (1927) found that apart from consumption of *khesari* pulse, in some cases *teora dal* was also responsible for the causation of lathyrism. Mackenzie (1927) observed that lathyrism attacked only people whose diet consisted of *L. sativus* grown on virgin soil or on newly cultivated land and the same plant after a few years' cultivation lost its lathyrogenic properties. Young (1927-28) examined the epidemiological and clinical aspects of human lathyrism in Central India. Stott (1930) experimented on ponies and fed them either *Vicia sativa* or *L. sativus* and found that these grains did not produce lathyrism. Stockman (1929) could reproduce in monkeys most of the symptoms

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of human lathyrism. Stockman (1932) maintained that all the legumes contain a poisonous substance capable of damaging the nervous system and that excessive consumption of any one of them could produce harmful effects. In Syria, Trabaud *et al* (1932) found that the epidemic of lathyrism which occurred during the 1914-18 war was due to eating *L. sativus*. Stockman (1934) thought that phytates, present in leguminous seeds, were mainly responsible for the production of lathyrism.

Mellanby (1934) considered lathyrism to be a deficiency disease. He claimed to produce the disease in dogs by feeding them on akta (*Vicia sativa*) and vitamin A-free diet. He thought that lathyrus contained a neurotoxin which became active in the absence of vitamin A or carotene. Basu *et al* (1937) opened that since the *L. sativus* proteins were deficient in tryptophan, all the symptoms of the disease might be due to the absence of this amino acid. Shah (1939) after observing the epidemic of lathyrism in the village Nawan Kulla in Narowal Tehsil in the Punjab, came to the conclusion that it was *Vicia sativa* which was responsible for the causation of the disease. Minchin (1940) found that lathyrism was produced without consumption of lathyrus. In Madras General Hospital, he saw 21 patients suffering from a syndrome of upper motor neurone paralysis affecting the legs, but occasionally ascending higher. Their diets did not contain *Lathyrus sativus* at all although the disease was identical with lathyrism.

Jimenez-Diaz (1941), Jimenez-Diaz and Vivanco (1942), Jimenez-Diaz *et al* (1942), Jimenez-Diaz *et al* (1943) conducted series of experiments and made an intensive study on the subject and declared that lathyrism was found in only those who ate lathyrus, that there was no reason to believe that deficiency of vitamins or amino acids was the cause of the disease, nor was there any proof to show that it was a virus disease. Ahmad (1944) thought that lathyrism was a deficiency disease. Ranjan (1944) observed dimness of vision in early cases of lathyrism. Shourie (1945) found that all his patients had taken *L. sativus* for 6 months or more, and there was no contamination of the grain with akta. Jacoby (1946, 1947) found that his patients had consumed *teora*, a species of the lathyrus genus. He observed that prostigmine had definite curative properties in the treatment of human lathyrism. Rudra and Bhattacharya (1946) noted that in human lathyrism, the serum alkaline phosphatase was greatly increased. This theory of pathogenesis of lathyrism was challenged by Chu *et al* (1948) who did not find significant changes in the concentration of alkaline phosphatase of the serum in rats in which experimental lathyrism was produced by feeding diets containing 50 per cent sweet pea (*L. odoratus*). Denny-Brown (1947) considered that it was by no means conclusively proved that human lathyrism had ever been experimentally produced or the disorder is produced by *L. sativus per se*. Vivanco and Jimenez-Diaz (1951) studied the condition produced in rats by feeding them with *L. odoratus* (sweet peas) and compared it with human lathyrism. In rats there was widespread involvement of bones, whereas human lathyrism was mainly a neurological trouble. In order to avoid confusion between the two types of lathyrism, they preferred to call lathyrism in rats odoratism. After experimenting on monkeys, Rudra (1952) noted that the toxic substance of *L. sativus* responsible for lathyrism was selenium. Rudra *et al* (1952) on an analysis of different foodgrains showed that lathyrus peas were poorest in methionine content and therefore the deficiency of this amino acid might be the cause of the disease.

In an attempt to produce neurolathyrism in animals, various workers tried drugs on mice and rats. Goldin *et al* (1948) found that dialkyl and heterocyclic-beta-chloroethylamines produced a 'waltzing syndrome' in mice. But Gruneberg (1943) had observed some normal varieties of mice which were either 'whirling, or circling or figure-eight dancing'. Rothlin and Cereletti (1952) noted that certain mice of their own laboratory strain also developed the waltzing anomaly spontaneously. Delay *et al* (1952) observed that aminodipropionitrile produced permanent motor overactivity in mice, and nicotinic acid potentiated this action. Thuillier *et al* (1953) opined that spontaneous spinning movements of mice described by Rothlin and Cereletti (1952) and the waltzing movements of mice produced by the administration of aminodipropionitrile, observed by Delay *et al* (1952), resembled closely. Bachhuber *et al* (1955) found that in rats, bis-β-(cyanoethyl)-amine has more pronounced effect on the central nervous system and less influence on the skeletal system. Azima and Grad (1956) noted that subcutaneous injection of β,β-iminodipropionitrile (IDPN) in mice produced a disturbance of functions of the central nervous system. Hartman and Stich (1957) observed that bis-β-aminopropionitrile (Bis BAPN) was a definite psychopathogenic compound.

All these compounds, however, could not elicit symptoms similar to those of lathyrism in man, nor has anyone been able to demonstrate the presence of any of the compounds, mentioned above, in the lathyrus responsible for causation of human lathyrism.

On the other hand, the history of osteolathyrism, also known as odoratism or experimental lathyrism, is a recent one. Geiger *et al* (1933), for the first time observed that rats fed on diets rich in *L. odoratus* developed deformities in the long bones, spinal curvature and enlargement of costochondral junctions. The animals became lame, their fur became sparse and lustreless and their growth was retarded. The long bones, on naked eye examination, showed various tumour-like formations, better known as exostoses. These observations were soon confirmed by Robinson and Bast (1934). To them, histologically, the exostoses appeared very much like callus formation in a bone fracture. Lewis and Esterer (1943) noted that large amounts of protein (casein) or ascorbic acid administered with the sweet pea meal diet did not prevent or delay the onset of lathyrism in the rat. Caley peas (*L. hirsutus*) when included to the extent of 25 to 50 per cent in otherwise adequate rations of rats, produced anatomical deformities characteristic of osteolathyrism (Turney *et al*, 1944). Besides *L. odoratus*, *L. hirsutus* and *L. tingitanus* also caused changes typical of osteolathyrism in rats (Lewis *et al*, 1948). These workers found that *L. sylvestris wagneri* was the most toxic. Lewis and Schulert (1949) observed that osteolathyrism could not be produced in young mice by feeding them diets containing *L. odoratus*. This finding was refuted by Dasler and Milliser (1957) who demonstrated that on sufficiently high levels of BAPN (active principle of *L. odoratus*), changes characteristic of osteolathyrism occurred in weanling male albino mice. McCallum (1958) extended the work of Dasler and Milliser (1957). That the ingestion of diets containing 70 per cent *L. pusillus* causes osteolathyrism in rats was observed by Lee (1950). When albino rats were on a diet containing 50 per cent sweet pea, they soon grew unhealthy and developed kyphoscoliosis and various types of bone deformities and some of them also suffered from aortic aneurysm. Ponseti and Baird (1952) concluded that sweet pea seeds had some toxic factor in them which affected the formation of organic bone matrix, the aorta and some mesodermal tissues. Extraordinary lesions were produced in the transitional zone of epiphyseal plates when diets containing sweet pea were fed to rats (Ponseti and Shepard, 1954). The epiphyseal plates became very wide and the cartilage matrix lost cohesion. As a consequence of these lesions, many defects developed in the experimental animals, viz., slipped epiphysis, kyphoscoliosis, degenerative arthritis, abdominal and inguinal hernias, diastasis of the sacro-iliac joints and dissecting aneurysms of the aorta (Ponseti *et al*, 1956). That the disease was not caused by deficiency of calcium or phosphorus, was proved by McKay *et al* (1954). Enlargement of the adrenal cortex in male lathyritic rats was noted by Dasler (1954) and Telka and Kuusisto (1956). From observation of the lesions in osteolathyrism, it appears that the tissues which are high in collagen content, namely skin, bone and blood vessels, are those which are primarily affected in the disease (Dasler, 1954).

Various workers attempted to isolate the toxic factor present in lathyrus genus seeds responsible for the causation of experimental lathyrism. Finally, it was Schilling and Strong (1954) who accomplished the isolation from *L. odoratus* seeds of a crystalline substance capable of producing the skeletal lesions characteristic of experimental lathyrism and it was concluded that the substance was β-(gamma-L-glutamyl)-aminopropionitrile. Soon Dasler (1955) and Wawzonek *et al* (1955) found out that the toxic properties resided in the β-aminopropionitrile portion of the molecule, the gamma glutamyl radicle being redundant. So the lathyrogenic compound came to be known as β-aminopropionitrile or just aminopropionitrile, briefly called β-APN or APN or BAPN. Chang *et al* (1954) found that in xenopus larvae, sweet pea feeding caused dislocations of the joints of the hind legs and loosening of the septi of the metameres of the dorsal musculature. Stamler (1955) observed that *L. odoratus* peas and certain aminonitrile compounds, when added to the diet of pregnant rats, caused death of the foetal rat late in the intra-uterine life. Pyörälä *et al* (1956) confirmed the observation of Stamler (1955). When pregnant rats were fed sweet peas from the day of conception, the embryo could not survive beyond the 16th day of gestation, and began to be resorbed (Walker and Wirtschafter, 1956b). Ponseti *et al* (1956) synthesised aminoacetonitrile with ^{14}C in its cyanide group and found that when this was administered to weanling rats, the major portion of the radioactivity was eliminated in the urine. Walker and Wirtschafter (1956a) considered that the dissecting aneurysm of aorta, which one found in lathyrus-fed rats, was just one manifestation of the generalised metabolic disorder of the elastic tissue. Lysis of elastic fibres and hyperplasia of fibroblasts were observed even in the pulmonary and coronary arteries. Walker (1957) also observed aneurysm in innominate, right and left carotids, right and left subclavians, coeliac axis, hepatic artery, superior mesenteric artery, both renal arteries and both internal iliac arteries. Clemmons and Angevine (1957) noted that injection of BAPN in suckling rats caused multiple fractures.

Some investigators tried to locate the site of lesion in osteolathyrism, whether it was in the

ground substance or in the collagen fibres. Bean and Ponseti (1955) thought that the fault was in the ground substance of the mesenchymal tissues, the glue which held the frame together. Menzies and Mills (1957) considered that the basic lesion was an accumulation of chondroitin sulphate with the general breakdown of supporting structures such as collagen fibres. Bell *et al* (1957) could not find any deviation from normal in ash or water content of bone in lathyritic rats. Pyörälä *et al* (1957) thought that it was due to a disturbance in the metabolism of mucopolysaccharides. Follis and Tousimis (1958) concluded that the defect was due to a failure of tropocollagen molecules to form collagen fibres. Castellani and Castellani (1958) found that there was diminution in the mucopolysaccharide content of ground substance in the case of osteolathyrism. Krikos and Orbison (1958) thought that the end-result of β-APN on wounds in rats was a disturbance in collagen formation. Gardner *et al* (1958) noted marked changes in the masticatory apparatus of osteolathyritic rats. They thought that the toxic factor of sweet peas caused destruction of the cementing substance of the mesenchymal tissues. Schwartz (1959) after experimenting on rabbits, concluded that there was an increased destruction or depolymerisation of the aortic ground substance in osteolathyrism.

Of late, much attention has been focussed over the study of osteolathyrism as every one of the disorders produced in rats by feeding them sweet peas has a counterpart in diseases in human beings affecting the mesenchymal tissues (Ponseti and Shepard, 1954). These diseases constitute a collection of pathological entities of unknown pathogenesis and unsatisfactory treatment. So if one could successfully tackle cases of experimental lathyrism in the laboratory, fresh vistas may be opened up for treating these diseases by using experimental lathyrism just as a model. The most important counterparts of osteolathyrism are Legg-Calvé-Perthes disease, Madelung's deformity, Paget's disease of the bone, infantile cortical hyperostosis, recurrent dislocation of the shoulder joints, adolescent kyphosis, osteogenesis imperfecta, etc.

Incidentally, it might be worth noting that there are some similarities between neuro- and osteolathyrism. In both types, it is the male who gets the greater brunt of the disease, the female coming next. It is known that in early cases of neurolathyrism if *L. sativus* is withdrawn from the diet, the patient might recover to his original state (Buchanan, 1904). The observations of Ramamurti and Taylor (1958) that discontinuation of APN from the diet of osteolathyritic rats was followed quickly by the condition of the animal to the norm shows that this is true of osteolathyrism also. However, no relationship between the two types of lathyrism has been established.

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JOURNAL OF THE
INDIAN MEDICAL ASSOCIATION

CALCUTTA, APRIL 1, 1961

ULCERATIVE COLITIS

Elsewhere in this issue is published an article on 'Management of Idiopathic Ulcerative Colitis'[1]. During recent years notable advances have been made in the diagnosis and treatment of bloody fluxes. Laboratory examinations and radiological techniques in gastroenterology have improved, while with the advent of sulphonamides bacillary dysentery has lost much of its dread. And yet despite these gratifying accomplishments one disease of the colon has remained singularly invulnerable to curative therapy. It is ulcerative colitis. This disease which is regarded as a problem for the physicians, surgeons and psychiatrists in the Western countries, is perhaps less often talked of or diagnosed in this country. While not disputing the relatively uncommon occurrence of this malady in this country, we feel that it may now be masquerading under various names such as, postdysenteric colitis, mucomembranous colitis, specific dysenteries—amoebic and bacillary, particularly the latter. It is well known that at times it becomes difficult to differentiate acute cases of idiopathic ulcerative colitis from acute bacillary dysentery, except by careful bacteriological examination of the stool. Similarly, when a given case of bloody diarrhoea fails to respond to adequate specific therapy, the possibility of ulcerative colitis as the cause for the diarrhoea cannot be definitely ruled out and appropriate investigations in that direction may often be rewarding. The actual incidence of this disease in this country is not known but it is likely to rise in view of the increase in the stress and strain of modern life, which is an important background in the precipitation of colitis. It is, however, not easy to determine whether the disease is psychosomatic or somatopsychic. The neurotic attitude of the patient with chronic ulcerative colitis is not undeniable. But then who could suffer from chronic diarrhoea and yet remain cheerful? Nevertheless, there is evidence to incriminate the psychogenic factor in the pathogenesis of ulcerative colitis. The significance of this malady to the afflicted individual is distressing and it invariably leads him to chronic invalidism. The danger to life is also not insignificant; for the M.R.C. trial[2], 11·9 per cent of patients died during the six weeks' observation. In another series[3], 46 per cent of cases was reported dead in the first attack. This dismal picture is no doubt due to lack of specific therapy for the disease. However, during the past few years improvement in both medical and surgical care has brought more hope to these unfortunate victims. The present article[1] is a pointer that much can be done for these patients to alleviate their sufferings and help them to lead near-normal lives. Hospitalisation is essential for fulminant and acute cases. This provides a change in the environment as well as rest, both physical and mental. Phenobarbitone in small doses (grain ½, thrice daily) may be necessary for procuring mental calm. Astringents and adsorbents are practically useless. Mild laxatives like small doses of sodium sulphate may be more useful than astringents. Broad spectrum

[1] CHAUDHURI, R. N., CHHETRI, M. K. AND CHATTERJEE, S. B.—*J. Indian M. A.*, **36**: 277, 1961.

[2] Editorial—*Brit. M. J.*, **2**: 1479, 1957.

[3] DEMOLE, M.—*Ibid*, **2**: 236, 1957.
Idem—*Ibid*, **2**: 1041, 1947.

antibiotics should not be used indiscriminately, for they may aggravate the condition by producing resistant strains of bacteria in the gut with their usual complications. Mild or moderate cases will derive benefit from sulphaguanidine or similar preparations, perhaps by their action upon secondary invaders of the ulcerated colon. Supportive measures are often life-saving, and proper correction of water and electrolyte imbalance and timely blood transfusion are invaluable in many cases.

A new approach to the management of the condition has been provided by the use of steroids first reported by Truelove and Witts[4]. Cortisone, 100 to 300 mg. daily, was able to secure remission in 40 per cent of cases (particularly the first attack and relapsed phase) compared to about 16 per cent remission in the control series (Truelove and Witts, *loc. cit.*) It is heartening to note that using much smaller doses of steroid, Chaudhuri *et al*[1] were able to obtain striking improvement in 7 acute cases and partial improvement in 9 patients in the chronic phase of illness, in a total of 30 cases. One of the two patients who received hydrocortisone hemisuccinate sodium rectally also showed remarkable improvement. It may be mentioned here that several workers[5, 6, 7] have earlier reported favourably on this form of treatment.

Response to steroid therapy has been less notable in the chronic phase of illness and there has also been failures in such cases, a point which is clearly illustrated by the present paper[1]. Used in properly selected cases steroids can be of real help and in some cases of lasting benefit to patients to whom till recently we had nothing else to offer except surgery, perhaps in desperation. The standard operation of ileostomy combined with subtotal colectomy has its own drawback namely social handicap but still it appeals to many patients. The surgical mortality is about 5 per cent in the best hands[8]. Aylett's[9] operation of anastomosis of the ileum to the rectum to avoid a permanent ileostomy, has been claimed to be superior, but in practice this may not always be possible as the rectum is either invariably involved by the disease or is likely to be affected in future[10]. Two cases in the series reported here who had previously received steroid therapy were subjected to surgery but ultimately died. This confirms the view that pre-operative steroid therapy adds greatly to the operation risk.

Ulcerative colitis is still a problematic disease and the paper by Chaudhuri *et al*[1] has been timely. It is hoped that this will stimulate greater interest in the subject in this country also.

[4] TRUELOVE, S. C. AND WITTS, L. J.—*Ibid*, **2**: 375, 1954.
[5] TRUELOVE, S. C.—*Ibid*, **2**: 1267, 1956
Idem—*Ibid*, **1**: 1437, 1957.
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[6] NABARRO *et al*—*Ibid*, **2**: 272, 1957.
[7] WATKINSON, G.—*Ibid*, **2**: 1071, 1958.
[8] BROOKE, B. N.—*Lancet*, **2**: 532, 1956.
[9] AYLETT, S.—*Brit. M. J.*, **1**: 489, 1957.
[10] GOLIGHER, J. C.—*Gastroenterologia*, **36**: 718, 1956.

XV MADRAS STATE MEDICAL CONFERENCE
OOTACAMUND. APRIL 1. 1961

The 15th Madras State Medical Conference will be held on Saturday the 1st April 1961 at the Tamilagam (Arranmore Palace), Ootacamund, Nilgiris district, under the auspices of the Nilgiris Branch of I.M.A. The Conference will be inaugurated by Shri D. P. Karmarkar, Union Minister for Health, Government of India, and the inaugural session will be presided over by Shri M. A. Manickavelu, Minister for Health, Government of Madras. Dr. V. R. Thayumanaswami, Director of Medical Services, Madras, will inaugurate the Scientific Sessions.

GENERAL PRACTICE DIGEST

ALLERGY TO STINGING INSECTS : MANAGEMENT AND TREATMENT

Reports of sudden deaths and severe reactions to insect bites have appeared in the medical literature from time to time. With regard to the stings of insects, one should distinguish between a true allergic reaction and a reaction attributable only to the toxin injected into the individual. The toxin is probably an acetylcholine-like substance. When the honey-bee stings, the stinger with the venom sac is retained by the person stung. The bee is then brushed off and dies. It takes a bee 2 or 3 minutes to inject the whole amount of venom through its fine stinger. So the sooner the venom sac is taken off, the less will be the venom injected. But hornets, wasps, yellow-jackets, and bumblebees have non-barbed stingers and do not leave their venom sacs. They are thus capable of further stinging.

The types of sensitisation have been divided into 3 groups, viz., those following : (1) innocently by scales or dust from wings or body, (2) injection of venom through sting, and (3) instillation of salivery secretions. Certain persons, however, fail to react to the bite of a given insect. The autopsy findings in fatal bee and wasp cases reveal oedema and emphysema of the lungs, over-distension of the right side of the heart, splanchnic dilatation with hepatic engorgement, and petechial haemorrhages of the skin and mucous membrane.

Correct diagnosis with proper treatment is spectacular and often life-saving. Statistics on accidental deaths attributed to certain insect bites and stings are by no means accurate, for some deaths from insect stings are recorded as natural, 'causes unknown'. The severe reactions to insects are mainly attributed to the wasp and the bee. The symptoms of patients with histories of sensitivity to wasp, ants, hornets, bees, and mosquitoes vary form severe local reactions to loss of consciousness. There are certain common allergenic properties of the antigens.

In the investigation by the authors the sera from affected persons were injected into individuals who had never had any injections of insect antigen except those used in skin testing. The authors' technique was as follows :

1. Studies were made on individuals who were negative to insects by history and skin testing.

2. Into each of the three sites in a vertical row on the lateral aspect of each upper arm was injected 0·1 ml. intradermally of sensitive serum (to which had been added as a preservative, 1 :100 of its volume of 1 :100 merthiolate, making the final concentration of merthiolate 1 :10,000).

3. At the end of 48 hours, the top two sites of the right arm were tested with wasp antigen of 1 :100 dilution and the top two sites of the left arm with bee antigen of 1 :100 dilution. These same sites were tested daily on each succeeding day until a positive reaction was no longer obtained by the wasp or bee antigen in the top two sites of each arm,

4. When the top site revealed a negative reaction (i.e., evidence that antibodies were exhausted), the site 2 of the right arm was tested with bee antigen and site 2 of the left arm was tested with wasp antigen.

5. On the last day of the test, site 3 of the right arm was tested with wasp antigen, and site 3 of the left arm with bee antigen.

Management

Treatment is divided into (A) immediate, which may be life-saving, and (B) prophylactic or desensitisation therapy.

A. IMMEDIATE TREATMENT :

1. *Local*—
   - (a) Wet alkaline packs.
   - (b) Ammonia water.
   - (c) Antihistamine ointments.

2. *Systemic*—
   - (a) Tourniquet, if patient has been stung on the extremity.
   - (b) Adrenaline hydrochloride (1 :1000) solution, 2 to 3 minims, administered into the site of bite, and in some other part of the body. This is given subcutaneously or intravenously only. Not more than 3 minims should be injected in any one site, because adrenaline causes enough vasoconstriction so that it is not absorbed readily. If necessary, several small injections are more efficient.
   - (c) Antihistamine should be administered intravenously.
   - (d) Plasma therapy, if there is evidence of haemoconcentration.
   - (e) Corticotropic hormones may have to be given intravenously.

B. DESENSITISATION TREATMENT :

Species or genus specific antigen is preferable. If a barb is found in the lesion, the sting is almost certainly that of a bee. Often the patient is seen too late to determine what the offending insect was. In such cases, a mixture of bee and wasp antigen is given.

The antigen is made from whole adult insects that have been caught alive and quick frozen, and kept in that state until the antigen is extracted. Insects that have been dead for any length of time are not suitable because of the change in the protein of the insect.

Technique of skin testing—A patient with a history of severe insect sensitivity, especially to bee, wasp, ant or mosquito, should be routinely punch tested. The punch dilution is 1 :20 (weight by volume). The testing is done with several dilutions, starting from 0·02 ml. of 1 :1,000,000 dilution. If there is no reaction, the 1 :100,000 dilution is given, then the 1 :10,000 dilution, and finally the 1 :1,000 dilution. The end point of titration is a local reaction or a systemic reaction, whichever occurs first. The end point is used as the starting desensitisation dosage.

Desensitisation therapy—The treatment is started in the next weaker dilution than the determined end point. Hyposensitisation dosages are given twice a week for 6 treatments, then once a week. The usual starting dosage of the determined strength will be 0·05 ml. It is increased to 0·45 ml., at which point the next stronger dilution is given. This is continued at weekly intervals until a 1 :100 dilution is reached, then 0·45 ml. of a 1 :100 dilution is given. This dosage is continued at monthly intervals indefinitely.

—From an article by R. L. Etter, W. J. Raymer and R. H. Jackson—*Texas State J. Med.* **56** : 915, 1960.

CURRENT MEDICAL LITERATURE

Prophylactic Haemodialysis in Acute Renal Failure

TESCHAN, P. E., BAXTER, C. R., O'BRIEN, T. F., FREYHOF, J. N. AND HALL, W. H. (*Ann. Int. Med.*, **53** : 992, 1960) from the Renal Branch, U.S. Army Surgical Research Unit, Brooke Army Medical Centre, Fort Sam Houston, Texas, give in the following lines the summary of their observation on prophylactic haemodialysis in acute renal failure :

Prophylactic haemodialysis has been employed in the treatment of 15 patients with acute renal failure due to acute tubular necrosis (12), bilateral renal cortical necrosis (two), and poststreptococcal glomerulonephritis (one). Dialyses, usually lasting six hours each, were begun before clinical evidence of uraemia developed in each patient and/or before the nonprotein nitrogen reached 200 mg. per cent, and were repeated daily or often enough to maintain the nonprotein nitrogen below 150 mg. per cent. The hypothesis underlying this technique postulates (1) that wasting, sepsis and impaired wound healing in these patients may reflect tissue injury by the same dialysable toxic agents which produce the uraemic symptoms that are readily reversible by dialysis, and (2) that repeated dialyses should therefore prevent both clinical uraemia and the later, often lethal sequelae.

The results contrast dramatically with our own past experience in treating patients with acute renal failure with a carefully executed medical regimen together with haemodialysis on conventional indications. Except in one instance of crush injury with progressive intracerebral damage, and one brief occasion in another individual, these patients experienced a stable, convalescent clinical course, remained free of uraemic symptoms or chemical imbalances, ate at least three meals daily which were unrestricted in amount and composition, and were ambulatory between dialyses unless confined to bed by associated disease. Wounds healed well. Infection either did not occur, or subsided after appropriate therapy. Fluid restriction was liberalised by means of ultrafiltration with dialysis. Regional heparinisation of only the extracorporeal circuit eliminated actual or impending bleeding as a contraindication to dialysis. Chronic vessel cannulation made the frequent dialyses possible, but may have provided the route for repeated, transient bacterial contamination of the blood stream in the first hour of many dialyses. Marked anaemia, despite reticulocytosis, moderate to mild weight loss and some mental deficit persisted in spite of the general clinical improvement and well-being. Three patients with tubular necrosis died after 7, 11 and 26 days of oliguria; both patients with bilateral renal cortical necrosis also succumbed, on the seventy-third and ninety-second days of renal failure, and after 29 and 40 dialyses, respectively. At autopsy, evidence of sepsis was conspicuously absent. The remaining 10 patients survived. Thus some, but not all, clinical manifestations of acute renal failure appear to be favourably influenced by prophylactic dialysis treatment.

"Our initial experience in this group of 15 patients does not of course prove that freedom from complications and a significantly better outlook for survival can be assured to patients with acute renal failure by these methods. However, it seems to offer a reasonable hope of this possibility which we cannot attach to management by medical measures alone, or by dialysis on conventional indications. If this hope is realised in greatly extended, subsequent series, then it seems inevitable that some form of prophylactic dialysis, or some equally effective alternative, should be adopted in treating the majority of patients with acute renal failure.

Successful Management of Cobra Bite with Cryotherapy

MULLINS, J. F. AND NAYLOR, D. (*J.A.M.A.*, **174** : 1677, 1960) from the Department of Dermatology, University of Texas Medical Branch, Texas, give in the following lines the summary of their observations on the successful treatment of cobra-bite by cryotherapy :

Two important problems were highlighted by this case. First, and foremost, we were faced with the task of managing a very deadly snake bite without readily available antivenin. Life or death is seldom a significant issue in the pit viper bites common to the United States. Cobra bites, on the other hand, are attended by a very significant mortality when specific antiserum is not available. Death may occur in 8 to 12 hours from respiratory failure. Without antisera our problem was to prevent systemic absorption of the toxin in order to insure preservation of the boy's life. In our opinion, of the three ways to achieve this purpose, cryotherapy, amputation of the finger, and strangulating tourniquet, cryotherapy seemed most desirable because it offered some hope of preserving the patient's finger.

The correctness of our decision was borne out when no signs or symptoms of systemic toxicity developed. The saving of the proximal phalanx was also gratifying as this tissue would have been lost with either of the other two approaches.

Our experience indicates that cryotherapy is very useful for poisonous snake bites whether specific antivenin is available or not. We feel cryotherapy is practically mandatory when specific antiserum is contraindicated by pre-existing sensitivity to animal sera.

A second important problem brought out by this case is the absence of legislative control of snake handling in most states. It is our feeling that persons under 21 should not be allowed to possess or handle poisonous

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reptiles. We also feel that legislation should be enacted requiring those who handle snakes to have on the premises specific antivenin for each variety of poisonous reptile in their possession. Varieties for which antisera are not available should be made illegal for importation into this country until specific antisera are manufactured. If these rather simple reequirements were met, the incidence of envenomisation could be sharply curtailed, particularly that caused by the bite of exotic, extremely deadly species such as the cobra.

Oliguric Renal Failure of Surgical Origin

SHACKMAN, R., MILNE, M. D. AND STRUTHERS, N. W. (*Brit. M. J.*, **2**: 1473, 1960) from the Department of Surgery and Medicine, Postgraduate Medical School of London, Hammersmith Hospital, London, write:

Acute extrinsic oliguric renal failure (which includes pre-renal and post-renal obstructive oliguria) and acute intrinsic oliguric renal failure may complicate surgical and traumatic conditions.

Experience gained from the management of 106 such cases is presented: extrinsic renal failure was present in 27 (4 pre-renal and 23 post-renal) and intrinsic failure in 79.

Intrinsic renal failure was due to acute tubular necrosis in 77 cases and cortical necrosis in 2; it complicated surgery or surgical conditions in 50 and followed accidental trauma in 29.

The majority of the patients had beeen transferred at various stages of their illness, and were severe cases likely to require treatment by an artificial kidney. This probably explains the apparent low incidence of pre-renal extrinsic failure.

Post-renal obstruction was a complication of pelvic malignant disease, urinary calculi, retroperitoneal fibrosis urological tuberculosis, and surgical operations. Treatment by surgery, and oestrogens in cases of prostatic cancer, was successful in 15 of 24 such cases. Haemodialysis was not required.

Of 50 patients with acute intrinsic renal failure following surgery or surgical conditions, the predisposing cause was peritonitis in 25, hypotension in 14, and infection (other than peritonitis) in 7. Renal-vein thrombosis, total nephrectomy, and intimal stripping at aortography accounted for the other four cases. Haemodialysis, often multiple, was required in 25 of the 50 patients.

Hypotension was responsible for the acute intrinsic renal failure in the 29 post-traumatic cases. Road and vehicular accidents occurred in 18. Haemodialysis, usually multiple, was required in 16 of the 29 patients.

Success was achieved in only 34 of the 106 patients. The survival rate in the cases of acute intrinsic renal failure was only approximately 20 per cent, but a high proportion of the fatalities occurred in patients with non-lethal biochemical abnormalities: extrarenal lesions contributed significantly to the high mortality. Infection was the most serious hazard: serious complications included gas-gangrene, multiple fat embolism, gastrointestinal and intracranial haemorrhage.

Factors Affecting Acute Gastroduodenal Perforation

ROGERS, F. A. (*Surg. Gynec. & Obst.*, **111**: 771, 1960) from the Surgical Service of the College of Medical Evangelists School of Medicine, Los Angeles, writes:

An appraisal of a large group of patients with perforated gastric and duodenal ulcers has been presented in order to analyse various factors which separately and in combination affect the mortality rate. The significance of advanced age, the female sex, prolonged duration of the perforation, large perforations, concomitant serious disease in other systems, the incidence of gastric perforations, shock, and erroneous diagnoses have been cited for their particularly adverse effects. The high mortality associated with postoperative complications and concurrent hemorrhage was also noted. Pitfalls in operative management were described.

Tabulated results under each of these categories for 1,000 cases of gastroduodenal perforation occurring on a general surgical service from 1944 through 1958 are used to emphasise the importance of these factors. Suggestions for improvements in both supportive and operative treatment have been made.

Postoperative Massive Collapse of the Lungs

RAFFENSPERGER, J. G., DIFFENBAUGH, W. G. AND STROHL, R. L. (*J.A.M.A.*, **174**: 1386, 1960) from the Department of Surgery, Cook County Hospital, Chicago from an analysis of two patients who had early postoperative massive collapse of the lungs report that postmortem examinations in these patients failed to reveal the usual causes of atelectasis. Specifically, there were no bronchial secretions or obstruction, nor was there pulmonary compression.

The authors feel that the combined factors of prolonged anaesthesia with depressing drugs, the rapid absorption of highly diffusible gases at the termination of the anaesthesia, and reflex bronchospasm combined to produce this syndrome. Suggestions for the prevention of this catastrophe include more vigorous efforts to prevent vagal reflexes during surgery and hyperventilation with room air to displace more rapidly absorbed gas from the lungs. Finally, mechanical respiratory assistance will be necessary in selected patients who exhibit symptoms of hypoventilation in the period immediately after operation.

Preoperative Fluid Electrolyte Balance and Blood Volume

MANSBERGER, A. R. JR., (*J.A.M.A.*, **174**: 1941, 1960) from the University of Maryland School of Medicine, Baltimore, writes:

Preoperative correction of water and electrolyte imbalance must be based on the diagnosis of a specific derangement. Intravenous solutions are medicaments and should not be ordered unless the physician is fully aware of their exact solute content and of the effects to be anticipated from injecting them. Both clinical observation and laboratory data are essential to the recognition of water depletion, water intoxication, hyper- and hyponatraemia, hyper- and hypokalaemia, magnesium deficit, hypoproteinaemia, and abnormalities of plasma

volume and red cell mass. Attention to details of diagnosis and correction has widened the scope of surgical procedures and has improved the prognosis for patients classified as poor risks or elderly citizens.

Dupuytren's Contracture and its Surgical Treatment

HEYSE, L. W. E. (*J.A.M.A.*, **174** : 1945, 1960) from the Orthopaedic and Surgical Services, Elyria Memorial, Crile Veterans Administration, and Western Reserve University hospitals, Cleveland, writes :

The anatomy, pathology, and various theories of aetiology of Dupuytren's contracture are presented. Associated lesions and differential diagnoses and their importance are discussed. The various non-surgical methods of treatment of the past and present are reviewed and noted generally to be unsuccessful.

A comparison of the 3 main surgical approaches to the subject is made. Fasciotomies are found generally to be an incomplete or adjunctive treatment to be used in association with more major procedures especially in releasing major contractures prior to definitive surgery. Fasciectomy is firmly entrenched as the treatment of choice of many eminent surgeons. The results are usually quite successful but with the major drawbacks of a long convalescence being required and that when a poor result occurs a virtually functionless and unsalvageable hand is obtained. Plastic skin coverage procedures are often found to be necessary. Local resection through short longitudinal incisions is found to give satisfactory results with short convalescence and rapid resumption of normal function and normal activities. Recurrences can be expected to be low if properly performed and complications are infrequent. A clinical study of this procedure with follow-ups of one to five years is presented.

Packed Red Cells *versus* Whole Blood in Presence of Impaired Cardiac Reserve

DALTON, M. L. JR., TYLER, H. AND HARDY, J. D. (*Surg. Gynec. & Obst.*, **111** : 732, 1960) from the Department of Surgery, University of Mississippi Medical Centre, Jackson, write :

Arteriovenous fistulas 1 centimetre in length were created between the abdominal aorta and the inferior vena cava midway between the renal vessels and the aortic bifurcation in 35 dogs, 20 of which reached the chronic phase of cardiac decompensation.

Once chronic cardiac decompensation had developed, the haemodynamic effects of a transfusion of 1,000 millilitres of whole blood and 500 millilitres of packed red cells were compared by various criteria.

The right atrial pressure recording is considered to be a most sensitive gauge of impending "overloading of the circulation", and by this and other criteria a volume of 500 millilitres of packed red cells was shown to produce less of a physiologic insult than an equivalent amount, 1,000 millilitres, of whole blood.

It is concluded that, in the presence of serious impairment of the cardiac reserve, the replacement of red cell mass deficit with an infusion of packed red cells imposes less cardiac strain than would the same volume of red cell replacement using whole blood.

CURRENT TOPIC

XLVIII INDIAN SCIENCE CONGRESS

Roorkee, January 3-9, 1961

The forty-eighth Session of the Indian Science Congress was held on 3-9 January, 1961, in the Roorkee University Campus. The Session was inaugurated by Dr. Rajendra Prasad, the President of India, and presided over by Professor Nilratan Dhar, Director of Sheila Dhar Institute of Soil Science, Allahabad.

As in other years, the deliberations of the Congress were held under thirteen different sections. In addition to the reading of original papers, symposia and discussions were organised in collaboration with different sections in which scientists of various disciplines took active part. Popular lectures by outstanding Indian and foreign scientists were also an attractive part of the Congress.

The Section of Medical and Veterinary Sciences was presided over by Dr. A. K. Hazra, Assistant Director of Haffkine Institute, Bombay, with Dr. Kalyan Bagchi, Associate Professor of Biochemistry and Nutrition of the All-India Institute of Hygiene and Public Health, Calcutta, as Recorder. About 80 original and interesting papers on various aspects of medical and veterinary sciences were presented before the Section, followed by lively discussions. Another interesting feature of the Section was the holding of several symposia in collaboration with other Sections. The symposium on "Diet and Fertility" held in collaboration with the Sections of Physiology, Agricultural Sciences and Statistics evoked keen interest among scientists of many disciplines. The consensus of opinion was that no definite evidences are available to show that over-fertility are associated with malnutrition or rice diet increases fertility. On the other hand, scientific experiments show that undernutrition or nutritional deficiency interferes with normal reproductive cycle as is evident in experimental animals. Recent census figures in India show that increased birth rates and child-mother ratio are found in areas or regions in India where the over-all nutritional and dietary status is much above the average.

The symposium on The Scope and Facilities for Medical Research in India also attracted a large number of scientists who participated in the discussion. The symposium on Recent Advances in Virus Researches in India covered a very important field, both in the domain of medical and veterinary sciences and the discussion included various aspects of chemotherapy in which workers from different centres evinced interest.

The next Session of the Indian Science Congress will be held in January, 1962, at Cuttack. Dr. J. C. Banerjea, Director-Professor of Medicine, Medical College, Calcutta, has been elected Sectional President and Shree Seetharaman of the Indian Veterinary Research Institute, Izatnagar, as Recorder of the Section of Medical and Veterinary Sciences.

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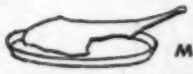

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PRESIDENTIAL ADDRESS*

I like to put forth some thoughts that have occurred to me about Medical and Veterinary Sciences, during my work in the line all these years. I will not like to call this an Address, but rather a communion with you on what we all feel about Medical Science as a whole. My colleagues will agree that every progress presents a challenge in itself. Hence I prefer to call today's talk "Progress—A Challenge".

I feel we should devote this time to stock-taking of what has been achieved during the last 10 to 12 years in our country after attainment of independence. Looking back at the history of Medical Research in India, we find that a base was laid by the Medical Research Council of India which gave an impetus to medical research by providing opportunities to young talents having aptitude for medical research. The emphasis in the olden days was mainly on teaching. The field of research was not stressed. But during the last decade, important changes and advancements have been taking place in various aspects of the Sciences, such as Teaching and Research, both applied and fundamental. I, therefore, consider that we must look at the problem in a broader perspective keeping in view India's development as a whole. What is the role of Scientific Research in developing the country's resources? Today we see Western countries making great progress in these sciences. We see the tremendous success they have achieved in Atomics and now in Space Travel. Our subject today is a different one but what I want to stress is that Science is the fundamental basis which is important for the development of our country. Science plays a very important role in harnessing the resources of an under-developed country like ours and that is why we should feel more concerned about it. Medicine is one branch of such science dealing with the health of the individuals and society alike, with which this Section is concerned today. I propose to deal with the subject under the four main aspects: (1) Teaching; (2) Medical research; (3) Providing adequate medical relief; and (4) Production of essential drugs in the country.

TEACHING—MEDICAL EDUCATION:

To begin with, it would be proper to consider the advances and changes effected in our teaching during the last decade. Medical research and education should go hand in hand—one stimulates and strengthens the other. We cannot make water-tight compartments of these two. In the past probably, the main purpose of medical education was not quite the same as it is at present in view of our present-day changing problems and newer approaches to the problems. These changing requirements for our medical needs were realised by those who are responsible for shaping the medical education. This realisation resulted in liberal experiments and suitable modification, etc., in various medical curricula. No doubt some of these changing aspects led to decisions not unusually acclaimed as beneficial for the aims in view. To mention a few, the question of medium of education as well as the contemplated reintroduction of the L.C.P.S. Course. Both these have been rightly subjects of controversial criticism.

I feel that emphasis should be laid on training of personnel in medical practice, medical research and medical administration, as these form the backbone of overall development of medical education in the country. Research can only grow and be profitable through proper medical education in a country and realistic appreciation of our problem.

I am afraid I have my limitations in saying anything authoritatively about medical education. I would, therefore, like to leave this subject to more experienced colleagues of mine in this line and with this view in mind a Symposium has been arranged where this specialised subject would be more fully and adequately discussed, in the light of the experiences of experts in various branches who have assembled here. Incidentally, I may bring to your notice that when an enquiry into the state of medical education in India was initiated by the Medical Council of India in 1953, almost every university reported a decline in medical education, both in standard and in teaching. This opinion tells us how big a problem we have to tackle in improving our medical education.

I feel it appropriate here to make a passing reference regarding the "medium of instruction" which is a burning topic largely discussed at State levels. A language is a medium of communication of ideas, thoughts and facts and should never be looked upon with racial and provincial attitude, or with any type of fanaticism. Some States in the country are feverishly thinking of adopting immediately the regional language as the medium of instruction in teaching of medical sciences. How far practical this idea is, is a matter for grave consideration from the point of view of all the students getting instructed in the subject in the best way possible. English has been the medium of instruction for the past two centuries or so in this country. We fully know that most of the world literature in Science today is available in English. As a matter of fact, English today can be considered as the International Language for technical subjects. Science and Medical Sciences are truly international in scope and advancement. Though the national languages are adopted in other countries as medium of instruction, the problem in India is peculiar because of the prevailing conditions. There are innumerable dialects in our country and more than a score of regional languages. You can very well imagine the confusion in adopting more than a dozen various languages as media of instruction in a single country. A student studying in one State may choose his career in a different one or go on changing these when taking up the All-India Service. Introduction of regional languages would mean publication of text-books, journals and other related information in many different languages in India. Available and useful scientific literature in English at the present moment is so vast that it will be impossible to cope up with translations in regional languages. All these would mean wasted effort. Science can grow and advance only on international level and if we adopt re-

* By Dr. A. K. Hazra, President, Section of Medical and Veterinary Sciences, Forty-Eighth Indian Science Congress, Roorkee, 1961.

gional languages as medium of instruction our growth will be stunted as our medical and veterinary graduates would suffer from difficulty in communication and exchange of knowledge. This would lead to narrow provincial feelings, create vested interest and act as a brake in the progress of medical science itself. We should not view with alien attitude any language whether it is English or Hindi. We should take a practical view of things and it looks appropriate that English should be retained as medium of instruction in higher education till we are fully equipped with text books and other materials available in one accepted national language and not hamper our development. Whether we like it or not, English is one of the important international languages, all those interested in sciences will have to learn if we do not wish to be left back in progress.

Equally important is the question of syllabus for medical education.

To my mind reintroduction of L.C.P.S. Course with all admitted label of "inferior grade" can but lead to trends that may better be avoided. No hasty step need be taken.

It would be worthwhile considering the other important issue of the various systems of healing science. Ayurveda is the most ancient system of scientific medicine in our country which gave birth to the present scientific medicine. This branch was, in some past, so much advanced in medicine and surgery that it has taken place of healing science in the country. Ayurveda and other indigenous systems have remained as they were. These systems should also be developed and merged into one board of scientific medicine, so that all indigenous remedies could be put to scientific tests. There is a bias against each of these systems which is coming in the way in their fusion with one another. No one can deny that Ayurveda has treasures in it, if one can harness them properly and make use of them for alleviation of human suffering. The indigenous systems suffer from one fault, and that is, in making untenable claims for cures without showing any scientific bias for it. To quote one—the various remedies claimed for snake-bite treatment with which I have some personal knowledge. Large number of such claims have been tested in our laboratories only to find that they failed in one and all cases to prove the claims made. This is because they have been keeping these remedies as traditional cures without getting them scientifically investigated. It is, therefore, felt that unless there is co-ordination and fusion of all the existing systems of medicine, scientific medicine will not develop on all sides and there will be disharmony in the health programme of the country. In considering the various indigenous systems we should be guided by scientific facts rather than by emotions or expediency which will go against them in the long run.

Teaching in veterinary sciences, especially post-graduate teaching, needs more attention. Facilities for post-graduate training do not exist on extensive scale as they do in the case of medical sciences.

MEDICAL RELIEF :

Next to medical education is the important question of Medical and Veterinary Services. It is a very well-known fact that medical services in rural areas are far short of bare minimum of facilities. The majority of India's population is scattered in rural areas. The problem of medical relief, therefore, is to be tackled taking into consideration the rural population. The population in India is growing fast. According to the census authorities, the number is expected to reach the level of 52 crores by 1981. Such a rapidly increasing population raises vast problems and generally overtakes all normal rate of development. If we see the distribution of medical men in India, on one side we see principal cities choked with medical practitioners struggling to make a living—whereas in the villages there is hardly one medical man available for thousands of people. Reasons for this are not difficult to understand. In spite of efforts made up to now by the Government and all concerned, we have not been able to make rural medical practice attractive for fresh graduates, because of many glaring disadvantages and defects still existing in the rural areas. If we want to attract young medical practitioners to rural areas it is very necessary that the all important question of their remuneration should be adequately settled and, as a matter of fact, remunerations should be more than in the cities. This may serve as an incentive and compensate for the rural living conditions. The fact remains that rural conditions, at least for some time to come, are after all going to be the same, however much improvement we make in them. Unless the social and living conditions improve to an appreciable degree, the settling of young medical graduates in rural areas is going to be a difficult problem.

Some years back Government had a scheme of subsidiary rural medical relief whereby Ayurvedic graduates and licentiates were selected to run the medical relief work in rural areas. However, the scheme did not meet with success because of the above mentioned drawbacks in rural conditions.

In villages we find many vernacular educated persons who can be made use of in rural medical relief scheme, as these persons are settled in the villages and they will stick on to the villages. A system should be evolved to make simple remedies available to the rural population through such an educated person. The minor ailments, such as influenza, malaria, dysentery, general debility, etc., can be easily treated by such a trained worker. The administration of medicine can also be simplified by adopting some code word arrangement and giving preliminary instruction to the person concerned (who could even be a vernacular final) to diagnose simple ailments. An organised system of rural medical relief on the above lines will serve a very useful purpose and solve our medical relief problem to a great extent. Along with this we should try to settle young graduates in the villages by providing them adequate facilities, such as provision for a refresher course, so that the person does not feel that he is left behind in medical knowledge as compared to his colleagues in the city. It is also necessary that for a group of villages a small clinic with few beds, screening facilities, laboratory facilities, etc., should be available and not merely a dispensary. This will also provide the young medical graduate with adequate professional atmosphere where he can reasonably expect to

do better type of work. These clinics should be linked up with larger centres where further facilities may be available to the practising physician for referring his more complicated cases for advanced treatment. In short, we have to establish a link of trained local personnel of villages for rural medical relief; young medical graduates to be in charge of a clinic for group of villages and the clinic to be linked up with district hospital. Rural medical relief can only be a success if we can put into operation the above scheme. Forcing young graduates to villages will never solve this problem, on the contrary, it may make matters worse. The State Governments should, therefore, evolve a scheme broadly mentioned above in consultation with their public health and medical authorities to create a proper atmosphere for young aspirants to take up the much needed work of medical relief where it is most needed.

Another eqnally important aspect of rural health is consideration of control of communicable diseases and improvement of environmental hygiene to make people healthier. Government has done a lot with the help of many international organisations, such as W.H.O., Ford Foundation, UNICEF, etc., towards eradication of important diseases like malaria, leprosy, smallpox, cholera, etc. Still this leaves a vast area to be covered. I am specially referring to the increased incidence of filariasis, helmenthiasis, tuberculosis and leprosy which should demand a very serious consideration from authorities concerned. The nutrition of the people needs to be improved in the existing conditions. We should bear in mind that it is not only the mortality which figures highly in these disease control measures, but far more important is the morbidity associated with these diseases. The amount of human misery, loss of working hours, and economic loss to the country as a result of this, due to those chronic ailments, if calculated, would be staggering. Adequate prophylactic and preventive measures in addition to curative treatment should receive due priority. This could be made possible if adequate correct statistical data are collected regarding morbidity in these diseases. Arrangements should also be made available for extensive field trials with newer drugs which are available for prevention and treatment of these diseases. To achieve this, medical and public health departments have to work hand in hand. Efforts in this direction, if adequately coordinated, will decrease failures in prevention and treatment of above mentioned diseases. Rehabilitation programme for the handicapped is another important and useful aspect which needs more attention and consideration than what has been paid so far.

MEDICAL RESEARCH :

There are two aspects in Research; one is applied research and another fundamental research. Applied research plays a major role in building up the country's technological progress, where we could train large number of qualified personnel. In fundamental research, the number of persons who have competence and aptitude is very small, but this forms a solid base for applied research. That is why both these aspects of science are interdependent and they need to be developed.

Researches in the medical and veterinary sciences have assumed an important role since independence. Much is being done to foster research both in research institutions and also in teaching institutions. Financial aid for these is being made available through recognised bodies such as I.C.M.R., C.S.I.R., I.C.A.R., etc., and also from international agencies, viz., W.H.O., Ford Foundation Colombo Plan, Rockefeller Foundation, UNICEF.

Very often the word "Research" is used as a pass word for reduplicating what has already been done elsewhere, probably because in many places in our country, progress is measured by or importance is given only to, the quantity of work, viz., number of papers published—rather than the quality, importance or originality of work. Very often all the research efforts of an individual are directed to obtain merely higher postgraduate degrees. It is rather difficult to blame the individuals for this, because, getting higher positions in our laboratory unfortunately still depends often on the number of academic qualifications obtained and the number of papers published anywhere. The net result of this would be the encouragement of adventurers and the elimination of really serious and capable people who would like to attack difficult and worthwhile problems.

I would like to say a word about Research Schemes sponsored by the above recognised bodies at various institutions. A body of experts in various specialities should carefully screen the proposed research scheme and select suitable persons to conduct these researches and once such screening is done a fair amount of freedom of work should be given to the worker so that he could conduct his researches in a congenial atmosphere with freedom of thought. As far as possible the routine administrative red-tapism should be curtailed in such schemes. Periodical reports are insisted on from such workers by these bodies, which I feel should be curtailed as it serves no useful purpose. The point is, to see how far the progress is made by the worker and whether the funds allotted are usefully utilised. Assessment of results could be done at short intervals with the help of experts in the line. A body of such senior persons could go round the various institutions in which these workers are working and discuss the problems with them and give the necessary guidance and encouragement on the spot. This practice of stock-taking, guiding and encouraging research workers on the spot by experts in various branches of medical research is, to my mind, much more beneficial than merely calling for reports from research fellows working in various centres in the country from their working laboratories and judge their standard of work and decide the destiny of the research and the researcher.

By adopting the new procedure suggested, the country can utilise the knowledge and mature experience of experts to the best advantage, as proper impetus will be given to the right kind of research and waste of funds on second-rate research schemes will be avoided.

Another important thing would be to set up an organisation in the field of applied research in various productive centres, and for this matter, Government institutions engaged in research activities as well as productive work, be organised on an ideal footing for other

commercial institutions to follow. It is painful to note now that many institutions which were held in high esteem and had made valuable contributions in the past, have either, for reasons of inadequate personnel or lack of incentive for research, dwindled into mediocre "paper-producing" bodies or devoting mainly to production activities and taking the role of pedagogues by exploiting available recognition for post-graduate training facilities to produce the academic post-graduates who also turn out to be mediocre in their standard. This does not make any substantial contribution towards the medical education or relief programme at large. As it is, you know, the present generation, for economic reasons, strain more for academic degrees (which may be right in a way as I have said earlier), and not so much for practical achievements in laboratory. It will be fatal to the country's interest in this formative stage to lay more stress on degree hunting in research organisations. A balanced view should be taken of these things. There are medical colleges and post-graduate institutions in the country which will look after this aspect more appropriately and we should leave this activity to them rather than take initiative in these academic activities which ultimately hampers the progressive development of research in practical application.

There is no doubt a keen dearth felt in various fields of suitable medical and veterinary qualified personnel. Some concrete steps will have to be taken to reorientate medical curriculum with a view to attract best talents in research fields.

The beginning will have to be made to search out people best suited for the job. This will have to begin at an under-graduate level. Students with aptitude for research should be spotted out and encouraged by giving facilities for working on some research projects during their vacations. Such programmes are in vogue abroad. Very often a student while working on such a project takes a liking to a particular subject and chooses to work in that branch for higher studies. This, of course, necessitates presence of active departments in Medical and Veterinary Colleges, which by their research activities would be able to stimulate the imagination of young students. An argument may be put forward against this, in the sense that what results can be achieved on problems undertaken under this Vacation programme of research. Well, we do not expect quick results from any research project, but this programme is specially suggested with a view to exploit the available talent in our institutions and spot out talents, and once they are found, long-term research projects will automatically be handled by them bearing fruitful results in course of time.

It is also very necessary that there should be a good co-ordination between different workers engaged on research problems. It often happens that a chemist synthesises many drugs which crowd his shelves without adequate facilities for screening and at the same time there are many workers whose work is hampered because of lack of adequate help from the chemist. This holds good in almost all the disciplines of medical and veterinary sciences. Such a situation can be avoided by having a central organisation which could help in co-ordinating research carried out at different centres. This centre should also decide priorities of research problems to be undertaken in the country. In a way the Experts concerned with various research institutions to examine schemes could form part of a body in the Central Organisation and function on a more sound footing.

DRUG MANUFACTURE :

To harness all our resources, to raise the standards of living and create better conditions with regard to culture, industries, etc., the health of the people is an essential must. Besides nourishment, availability (and that too at all times if not wholly free at least at a cost within their means) of drugs that can cure common or serious illnesses which unfortunately still take a heavy toll of life and inflict ill-health, is important for the health of the people of our country.

At present except some quantity of antibiotics most of the important drugs are imported from abroad which drain our scanty foreign exchange. These imported drugs in finished form or bulk supplies are bottled in India and are sold at very exorbitant prices which are not within the reach of the common man (who is the main sufferer in this case). This problem therefore demands an immediate attention by all concerned. Fortunately, the solution lies within our reach, as the country has the required talent and man power, and with collaboration and co-operation of other neighbouring countries who have done enough spade work in developing drug industry, we can easily get the know-how to produce these drugs for our country and make them available at cheaper price which can be well within the reach of the common man.

India is an agricultural country and more care of our live-stock has to be taken. Development in animal husbandry is, therefore, very essential. The drug industry is based on chemicals, but one cannot deny that animals also are a very important ingredient in the development of drug industry. The other countries are developing in glandular products and they are putting to use waste materials, thus saving a lot of the country's resources. India too has enough of this material and it can learn many things from its neighbouring countries to develop this line. We have to lay stress on modernised slaughter-houses if we have to develop our glandular products project. In this, co-operation and co-ordination of scientists, administrators and Government and semi-government bodies is very essential. When this is available our country can easily make rapid progress in medical research and make available drugs for the benefit of the population at large. At the same time India can keep pace with the fast-growing development in various fields of medical research in other countries.

PATENTS :

In order that modern drugs be freely available, it would be necessary to develop drug manufacturing industries in our country with proper orientation of our policy to meet the situation. It is well-known that in private drug industries profit is the largest element in the sale-price—and this is safeguarded often by taking out patents which give a monopoly right. This undesir-

able tendency is fortunately realised by all interested in the development of drug industries in our country.

As you all know the Patents System in the country which exists for over 100 years was first introduced during the British regime to safeguard mainly interests of other than our country. It is a well-known fact that Patent Law is meant for the industrial advance of the country, and the monopoly right given to the inventor should not be used against the interests of the country or by keeping up the sale price of the article higher. As things are at present, the Patent Law, as far as food and drugs are concerned, is detrimental to the development of Food and Drug Industries, since a vast majority of patents are held by foreign patentees who continue to hold monopoly over those products and do not undertake their manufacture in the country, and thereby hinder the industrial progress of India in regard to drug industry as a whole.

The only alternative, therefore, it seems to me is to make the inventions relating to foods and drugs non-patentable, both as regards the product and the process of manufacture, as in the field of atomic energy. India has rightly taken steps in time to safeguard its progress in this field by making Patents Law non-operable in regard to atomic energy. This Section of Medical and Veterinary Sciences also strongly feels that the same should hold for food and drug industries in our country to ensure healthy progress. A suitable Resolution may, therefore, be adopted in this Congress re : Patents Law as far as this aspect of the question relating to Food and Drug is concerned. This is very vital as unless the hurdle of patents is crossed, our drugs industry will not develop freely and extensively.

With the prospects of applied research yielding itself in the fulfilment of self-sufficiency of various medical products, particularly, biological products for prophylaxis and therapeutics, both in medical and veterinary sciences, it is desirable that a very well-laid plan for the future regarding the Drug Industry in India should be drawn up with full considerations of available resources, requirements and targets.

Cheap and ready availability of essential drugs would be possible if we take care of the problem of Patent Law in the country. It is a good thing that Government is actively considering revision of Patent Law in view of the report of Justice Mr. Ayyangar. Government of India is going to invest about 30 crores in the Drug Project and in addition some States may also undertake to manufacture essential needs for tackling health problems. There should be no hindrance by way of patents as I said earlier, as there are no positive advantages to the country by the grant of patents.

NOTES AND NEWS

Achievements of the World Health Assembly

The 14th World Health Assembly opened in New Delhi on February 7 and ended on February 24, 1961. The President was Sir Arcot Lakshmanaswami Mudaliar, Vice-Chancellor of Madras University and Chief Delegate for India.

In his speech to the closing Session the President, Sir Arcot Lakshmanswami Mudaliar, said that delegates had shown that they had one goal, one ideal and one language. The goal was that of the World Health Organisation, the attainment by all peoples of the highest level of health. The ideal was that of promoting the well-being of mankind and the single voice was that of the medical profession, whose first duty had always been service to those in need irrespective of all other considerations.

Although certain matters which had occupied much of the Assembly's time might better have been discussed in another forum of the United Nations, the President emphasised that good solutions had been found to even the most thorny problems, and although during the debates differing viewpoints had been expressed forcibly, as they should be, delegates had accepted the decisions taken in a spirit of cordiality.

The Assembly had voted the largest budget the Organisation had ever had and had adopted a firm policy of eradication of disease. However, Sir Arcot appealed to the more favoured nations to continue their voluntary contributions for malaria eradication work. The need and the opportunity were now, he said, when so many newly-independent nations were emerging and needed help.

Referring to the Assembly's discussions on the health hazards of atomic radiation, the President appealed to all the countries concerned to listen in a proper spirit to the expressions of anxiety that had been heard from many delegates. He hoped that when they met again there would be a "clean slate" and the situation would be such that there would be no further need for illusions on this matter.

The Assembly was attended by delegates from WHO's 100 Member and Associate Member States, including those from one new Member, the Islamic Republic of Mauritania, and from two new Associate Members, Tanganyika and Ruanda Urundi.

The World Health Assembly adopted a programme of work for 1962, with a working budget of 23,607,180 dollars. This figure comprises the Director-General's original proposal of 20,852,000 dollars, plus several additions approved by the Assembly, including an amount of 2,000,000 dollars towards financing the field activities of the world-wide malaria eradication programme, 797,347 dollars for the administrative and operational services costs of the malaria eradication programme, 110,033 dollars for assistance to the Republic of the Congo (Leopoldville), and 21,400 dollars for the use of Russian as a working language in the European Region. A deduction of 203,000 dollars was made by the Director-General from his original proposal, following the decision of the General Assembly of the United Nations to reimburse WHO for the book value of its investment in the Palais des Nations in Geneva, WHO's present headquarters. A new headquarters for WHO is at present being built in Geneva.

The budget adopted for 1962 reflects attempts to preserve the continuity of WHO's evolution in recent years, with particular reference to the increasing importance of the stimulation and co-ordination of medical research;

the continued need to concentrate efforts on the world-wide eradication of malaria; the efforts to strengthen basic public health services and the wish to accept fully the responsibility which a very substantial increase in membership places upon the Organisation.

Twelve countries were elected to designate a person to serve on the WHO Executive Board. They are: Chile, Iceland, Iraq, Israel, Italy, Japan, Nigeria, Pakistan, Poland, Senegal, Spain and the United States of America. The twelve other members of the Board are from the following countries: Argentina, Ghana, Ireland, Jordan, Korea, Luxembourg, Nepal, Peru, Sudan, Thailand, United Kingdom and Venezuela.

Among the topics which received particular attention were:

Assistance to newly independent States—In its penultimate session the Assembly unanimously adopted a resolution concerning the granting of independence to colonial countries and peoples. Welcoming the attainment of independence by new States and their entry into WHO and stressing that WHO has an important part to play in promoting the right of colonial peoples to freedom and independence through assistance in raising levels of physical and mental health, the resolution appeals to the Member States of WHO to introduce or develop in their health education programmes the teaching of the principles of racial equality and non-discrimination with a view to promoting good mental health and in recognition of the fundamental right of every human being to health.

The Assembly also adopted a resolution voicing satisfaction with the speedy assistance given by WHO to the Congo and requesting the Director-General to give similar assistance to all newly independent States who become Members of WHO.

Malaria eradication—The Assembly approved a scheme to transfer gradually, over a period of three years, the cost of field operations in the world-wide malaria eradication campaign from the special voluntary fund to WHO's regular budget. Efforts to obtain voluntary contributions to the Malaria Eradication Special Account, from governments and private sources, would continue. It was also decided that, beginning in 1961, the administrative and operational services' cost of the malaria eradication programme should be financed from WHO's Regular Budget.

These measures were designed to ensure that the technical achievements in malaria eradication, with which the Assembly expressed satisfaction, would not be hampered by lack of funds.

Radiation health—A resolution defining the role of WHO in radiation health, including the protection of mankind from ionising radiation hazards, whatever their source, was adopted by the Assembly. The resolution notes that present scientific knowledge provides data on the harmful biologic and genetic effects on human beings of massive doses of ionising radiation, voices recognition of the anxiety of WHO Member States concerning increased exposure to ionising radiation and approval of the action of the United Nations General Assembly relating to the immediate cessation of nuclear test explosions.

Smallpox eradication—It was reported that the world-wide efforts to wipe out smallpox are making good progress and that most countries in which the disease is still present have now undertaken programmes to eradicate it. However, the Assembly adopted a resolution which noted that smallpox is still an important problem in international travel and that it was urgent to speed up eradication programmes.

The Assembly also gave approval to a resolution that WHO should have a flag, of the same blue colour as that of the United Nations but featuring the WHO emblem in white and gold. This emblem consists of the Aesculapian staff superimposed on the UN symbol.

The informal technical discussions this year dealt with "Recent Advances in Tuberculosis Control".

The Assembly decided that the Fifteenth Assembly should be held in Switzerland.

Uses and Abuses of X-Ray Examination

WHO Expert Committee on Tuberculosis felt strongly that chest x-ray examination by radiophotography or radiography is an extremely valuable tool in tuberculosis control and that the amount of radiation from properly constructed and properly operated mechines used for such purposes is negligible compared with the benefits when chest x-ray examination is indicated. Nevertheless, the Committee advocated that every possible effort should be made to abolish all unnecessary radiation; it believed that fluoroscopy should not be used as a screening procedure, not only because of the greatly increased degree of radiation but also because it does not provide a permanent record for comparison with subsequent x-ray films. Whether or not tuberculin testing should precede x-ray examination of the chest in various population groups to reduce exposure to radiation would depend on local circumstances (i.e., the prevalence of tuberculous infection and disease) and therefore should be determined locally; but the Committee concurred with the general view that the examination of children in countries where the yield of cases is known to be low in this age-group should be confined to those first shown to have been naturally infected (*WHO Chronicle*, **15**: 34, January, 1961).

Treatment of Radiation Injury

A Scientific Meeting on the Diagnosis and Treatment of Acute Radiation Injury was held in Geneva, from 17 to 21 October, 1960, under the joint auspices of WHO and the International Atomic Energy Agency (IAEA). It was attended by 30 leading scientists from France, India, the Netherlands, Poland, the United Kingdom, the U.S.A., the U.S.S.R., and Yugoslavia. The main topics discussed were: the acute radiation syndrome in man and its treatment; radiation given therapeutically and followed by tissue transplantation; and bone-marrow transplantation in man and animals.

While future radiation accidents may be of a different type from those experienced so far, the experts considered that certain features in the diagnosis and treatment of acute radiation injury will remain essentially unaltered. Clinical observation and acumen in both

diagnosis and prognosis will continue to be important, particularly in view of the difficulties often met in assessing the radiation doses received. Important factors influencing the pathological processes following accidental irradiation are the parts of the body exposed, as well as the type of radiation and the rate at which it is delivered. Blood tests, in the opinion of the experts, are important in diagnosis, though there may be considerable variations among the persons involved, even in a single accident. It was agreed that a strict medical regimen should be adopted immediately, and that treatment should be undertaken according to the symptoms and signs as they arise.

Treatment by antibiotics and blood transfusion was discussed, as well as the possibility of "replacement therapy" whereby, for example, damaged bone marrow might be replaced (*WHO Chronicle*, **15**: 33, January, 1961).

International Psychosomatic Cancer Study Group

The 2nd International Conference will be held from August 27th to 29th, 1961, in Paris. The theme of the conference will be "Psychosomatic Aspects of Neoplastic Disease". Particulars may be had from the U. K. representative, Dr. D. M. Kissen, Brookfield, Elm Walk, Bearsden, Glasgow, Scotland.

Discovery by U. S. Scientists

The mysterious substance which causes leukaemia (cancerous) blood cells to multiply rapidly and constantly has been identified by University of Washington scientists.

The American Cancer Society reports the substance is an enzyme, called DR, or Dihydrofolic Redutase. Scientists believe its identification may eventually help lead to a way to improve leukaemia patients' chances of survival.

DR works by setting in motion a series of chemical reactions which enable leukaemic cells, and possibly also very young normal cells, to multiply. When the substance is not present the cells cannot reproduce and they eventually die.

Scientists have known for some time that when certain drugs, such as aminopterin and amethopterin, are given to leukaemia patients there is often a remarkable improvement in their condition. But the improvement lasts only for several weeks or months, and the disease always recurs.

The University of Washington researchers found in DR the secret of the temporary improvement. They learnt that as the drugs destroy the more vulnerable leukaemic cells and the patient improves, the surviving cells and their progeny rapidly step up their production of DR until finally they produce enough to overcome the effect of the drugs. Then leukaemia again gets the upper hand.

The drugs cannot be given in doses large enough to destroy all the leukaemia cells because they are poisonous to some normal, fast-growing cells of the digestive tract and other organs.

The Washington researchers are Dr. Frank M. Huennekens, Dr. B. W. Gabrio and Dr. J. R. Bertino of the Departments of Biochemistry and Medicine.

They believe it is possible that new drugs of antisera (blood containing antibodies against DR) might be devised to knock out DR and improve leukaemia patients' chances of survival. The American Cancer Society, which supports their work says their findings may apply to other cancers as well as leukaemia.

Minimum Equipment for Medical Halls and Clinics

The Uttar Pradesh Medical Council has prescribed the minimum equipment, storage arrangement, furniture and books necessary for proper maintenance of dispensaries, clinics or medical halls when run by medical practitioners for their own patients. Medical practitioners who have their own clinics, etc., are requested to conform to the prescribed minimum. The list of prescribed equipment, etc., can be had free of charge from the Registrar, Medical Council, Uttar Pradesh, Lucknow. The list is as given below:

1. Premises—The premises should be kept quite neat and clean. As far as possible, the floor of the hall should be washable (either cemented or paved with stone slabs or bricks) and the walls should be plastered. Under present conditions it is not possible to prescribe definite floor area to be used but it is suggested that the floor area should be accommodative, so that it may be divided into three portions (a) one for reception, (b) for examination and consultation and (c) for dispensing, which should be separated by a screen or a barrier to prevent admission of the public.

In front of the clinic, the doctor's signboard should be of the prescribed size and, if possible, the name of the clinic may also be written.

2. Furniture—There should be requisite number of furniture and they should be, as far as possible, either painted white or varnished. There should be proper sitting arrangements for the patients. The dispensing table should be at least four feet by three (4′×3′) and it should be covered with proper oil cloth or should be made up of either stone or cement-concrete slabs properly polished.

Almirahs should be of ordinary size, with glass-paned doors and locks, painted either white or with some other sober colour. For poison, as far as possible, there should be a separate almirah and the word 'Poison' should be written in red on white background.

3. Apparatus—(a) Ordinary dispensing balance with apothecaries weight, (b) Glass stoppered bottles for water, (c) Bottles with droppers, amber or other coloured, assorted sizes, (d) Bottles for prescription, (e) Corks, assorted sizes, (f) Cork extractor, (g) Glass funnels, (h) Filter paper and gauze cloth, (i) Measure glasses of different sizes as need be, (j) Measure glasses cylindrical, assorted sizes, (k) Mortars and pestles, (l) Ointment pots, earthen, (m) Ointment slabs, porcelain, (n) Spatula, (o) Scissors, (p) Spirit lamp, (q) Glass rods and tubes of assorted sizes, (r) Thermometer, clinical, (s) Tripod stands, (t) Watch glasses.

4. Books—Indian Pharmocopoeia current edition and National Formulary of India.

CORRESPONDENCE

The Editor is not responsible for the views expressed by correspondents

Department of History of Medicine Osmania Medical College

SIR,—The Department of History of Medicine, Osmania Medical College, Hyderabad, is the only one of its kind in India, and amongst a few in the East. Besides giving lectures on the History of Medicine to the undergraduates and postgraduates, the department collects materials relating to the History of Medicine in India, of Ancient, Mediaeval and Modern periods, studies the materials already collected, prepares bibliographies and publishes the results of these researches for the benefit of the medical colleges and the universities in India and abroad. But this programme cannot be followed successfully without the large scale co-operation and help from various medical colleges and from the universities, which have departments of studies for Indian History, Indian Culture and Indian Literature, and from other learned institutions, as well as allied Government departments.

There are many requests for information on this subject from the different medical colleges in India and abroad. To meet the demand it is proposed to publish a bulletin of the Department of History of Medicine giving useful information on the study or teaching of History of Medicine in India and also periodically publishing lists of books for reference, articles for consultation, current bibliographies and new materials and sources for further study.

Personal attention is invited on the promotion and studies in History of Medicine in India from all persons interested in this subject. Any useful materials available in any institution which will be helpful in medico-historical studies will be gratefully acknowledged. Among the specific items of information or material which this department will welcome are the following:

(1) Lists of old and rare books, manuscripts, reports, records, pictures, portraits, relics, etc. relating to ancient, mediaeval and modern periods up to 1910.
(2) Gifts of old books, old journals, reprints of articles, photos of doctors, hospitals, staff, text-books and note books of students, letters of doctors of the last two centuries, instruments, containers and utensils used in the preparation of medicines, reports on the medical institutions, etc.
(3) Old journals of the various medical colleges and the reports of medical institutions and reports of addresses and reports of medical conferences.

The staff of the medical colleges and the universities interested in teaching and research in the History of Medicine in India are also welcome to correspond with the department, pay visits and utilise the facilities and make suggestions for expansion of our activities.

I hope that this letter will bring very encouraging response from the medical colleges and universities and learned institutions in India. I am, etc.,

Osmania Medical College, Hyderabad-Dn. A.P. D. V. SUBBA REDDY

REVIEWS

Emergencies in Medical Practice—Edited by C. Allan Birch, M.D., F.R.C.P. Sixth Edition 1960, E. & S. Livingstone, Ltd., Edinburgh and London; 8½"×5½", pp. xii+751; 45s. net.

The sixth edition of this popular handbook on medical emergencies has come out as a more useful treatise than its predecessor. A few changes have been made to convey up-to-date information. Emergencies concerning R.N.T. diseases have been grouped into one chapter; medical emergencies in air travel, hazards of radiation and gynaecological emergencies have been revised and rewritten with a definite improvement. The book has been written with the objective to assist the medical practitioners in the U.K., and hence the lists of special reference centres, therapeutic units or bodies with their addresses have been incorporated at proper places; specific medicolegal procedures have been laid down nicely Still no doubt, this volume will prove a worthy companion to the medical personnel elsewhere. The contributors have tried their best to do justice to their subjects by fairly covering the problems of emergency. They have also proceeded further to treat the patient after the crisis is over or the patient is hospitalised. Specialists in different branches of medicine like ophthalmology, otorhinolaryngology, obstetrics and gynaecology may also find interest in this book. Almost all the chapters are well written especially that on poisoning. Emergencies in tropics may seem to our practitioners not quite adequate, and there is no chapter on venereal diseases or sexual problems. However, the moderate size of the book might have prevented details.

Last but not the least, the beautiful get-up and a good number of fine illustrations have made the book all the more attractive.

BHUBAN SINHA

Surgical Convalescence—Published by the New York Academy of Sciences, Vol. 73, Art. 2, 9"×6", pp. 381-538, Sept. 10, 1958; $4·00.

It is a very useful book and will be helpful to surgeons, internists and senior medical students preparing for higher examinations.

S. BANERJEE

Antibiotics and Sulphonamides in Ophthalmology—By A. Sorsby and J. Ungar; 1960, Oxford University Press, London; 6½"×4", pp. 117; 12s. 6d.

In this small handbook Professor Sorsby and his co-author have dealt with all that is necessary to know about antibiotics and sulphonamides. The subject-matter has been dealt with in three parts. Part I describes the basic consideration about infection and treatment. Part II deals with the pharmacology and therapeutics of sulphonamides and a dozen antibiotics. Part III describes how the various ophthalmic diseases are to be treated with sulphonamides and specific antibiotics. All ophthalmic surgeons will undoubtedly be benefited by the wealth of knowledge it contains. The volume will also be useful to general physicians and medical students.

M. SENGUPTA

SUPPLEMENT

Journal of the Indian Medical Association

VOL. 36, NO. 7 CALCUTTA APRIL 1, 1961

BRANCH NOTES

ALLAHABAD BRANCH—On 16-12-60, a scientific meeting was held. Dr. Y. P. Naithani spoke on Management of Injuries in General Practice. On 16-12-60, the executive committee meeting was held, the programme for the year 1960-61 was chalked out. On 23-12-60, a scientific meeting was held. Dr. G. C. Agarwal spoke on Some Gleanings on Optical Fundus in General Practice. A number of cases with features of clinical interest was demonstrated. On 1-1-61, a festival cricket match was played and an At-Home arranged. On 8-1-61, the members had a subscription picnic cum lunch at Rambagh Gardens. On 12-1-61 the members arranged a dinner to the visiting doctors from U.P. and others. On 26-1-61 Republic Day was celebrated. On 26-1-61, the general meeting decided to open a Family Planning Clinic. Congratulations were sent to Dr. B. C. Roy and other doctors who received Republic Day Honours. On 30-1-61, Anti-Leprosy Day was observed in co-operation with the district authorities. A public meeting was held, presided over by Col. A. Nandi. The superintendent and medical officer I/C of "Mission to Lepers" Hospital, Naini, Allahabad and 15 leprosy patients attended the meeting and took great interest in the proceedings.

ANAMALLAI BRANCH—A clinical meeting of the branch was held on 20-8-60. Nine members were present. After the routine business was over, Dr. R. Harris gave a talk on the Anatomy of the Rectum and Anus and dwelt on some of the common allied conditions.

A clinical meeting was held on 17-9-60. Twelve members were present. Dr. Sivanandam, President, Madras State Branch, I.M.A., spoke on some of the pitfalls in medical practice. A sub-committee was formed to go into the question of having a building for the branch.

A clinical meeting was held on 22-10-60. Ten members were present. The members accorded welcome to Dr. Disawalla and Dr. (Mrs.) Disawalla on their return from foreign tour. Dr. Padmajan Pillai gave a talk on Nephritis. Dr. Disawalla and Dr. (Mrs.) Disawalla spoke on their impressions of the medical work and advances in U.K. and the continent.

A clinical meeting was held on 19-11-60. Fifteen members were present. The meeting welcomed Dr. P. Narayanan who had returned from foreign tour.

Dr. R. Perumal gave a talk on some conditions causing pain in throat. Films taken by Drs. Disawallas during their tour were shown.

BAREILLY BRANCH—The branch celebrated the Republic Day in an atmosphere befitting the occasion. About 200 persons enjoyed the programme: Cricket Match, Hockey Match, Tug of War, etc. The programme included a tea party in the evening and dinner at night.

A clinical meeting was held on 31-1-61. Dr. C. M. Aga presided. Dr. W. More, an American Gynaecologist, spoke on Acute Hepatitis in Pregnancy. Questions were asked by the members on the subject and were explained and discussed by the speaker.

BHOPAL BRANCH—A general meeting was held on 6-2-61 in the Lecture Hall of Gandhi Medical College. Fifty members were present. Dr. M. M. Arora presided.

Dr. M. L. Sharma, Chief Surgeon, Heavy Electricals, spoke on Experimental Studies in Air Embolism. Dr. M. Kumar, Professor of Surgery, demonstrated a case of Metastatic Hypernephroma in Nephrectomy Scar with Haematuria occurring 22 years after nephrectomy. Dr. M. S. Dwivedi showed some interesting x'ray plates of Hiatus Hernia, Volvulus of Stomach, Polyps in Duodenum. New office-bearers for 1961 are: Dr. M. M. Arora as president; Dr. R. P. Bhargava as vice-president; Dr. R. K. Bhargava as hony. secretary; Dr. K. Koshley as joint secretary; Dr. S. N. Sen Gupta as treasurer.

A general meeting of the branch was held on 20-2-61. Fifty doctors attended the meeting. Dr. M. M. Arora presided. The meeting was followed by dinner.

Dr. S. S. Gupta, Professor of Pharmacology, Gandhi Medical College, Bhopal, read a paper on "Some Observations on indigenous anti-diabetic drugs". He carried out experimental studies on albino rats and observed the effect of 'Gurmar" and "Tribhangshila" on normal and hyperglycaemic rats (rendered hyperglycaemic by anterior pituitary extract.)

Dr. M. P. Shrivastava, presented a case of Myeloid leukaemia with a bony tumour of left 9th rib with the radiological diagnosis of Osteoclastoma. Dr. S. M. Misra, Professor of Medicine, elaborated the case. In the absence of Biopsy of Tumour opinion was divided between Myeloid Leukaemia with Leukaemic Infiltration in the rib or bony tumour with leukaemoid reaction.

BHUBANESWAR BRANCH—The annual general meeting of the branch was held on 3-1-61. Dr. G. C. Pattanaik presided. Fifteen members were present. Office-bearers for 1960-61 were elected with Dr. D. Satpathy as president; Dr. G. D. Mahapatra as vice-president; Dr. K. C. Roy as secretary; Dr. Leelabati Pattanaik as jt. secretary; and Dr. U. C. Ghosh as treasurer.

BIJNOR BRANCH—In a meeting of the branch held on 7-1-61 the members condoled the sad and sudden death of Dr. (Mrs.) B. A. Singh. A copy of the condolence was sent to the members of the bereaved family.

CALCUTTA BRANCH—The executive committee of the branch in a meeting held on 10-1-61, expressed profound sorrow on the untimely death of Dr. Mrityunjoy Sen, an active member of the branch. The meeting conveyed sincerest condolence to the bereaved family.

CHITRADURGA BRANCH—The monthly meeting of the branch was held on 4-12-60. Forty doctors were present. The president, secretaries of Shimoga and Davangere branches attended the meeting on invitation. The conspicuous feature of the meeting was the inauguration of medical check-up programme. About 200 people of the surrounding areas were examined. The response from the public was encouraging. Dr. C. S. Narahari spoke on Family Planning.

In the evening a clinical meeting was held. Dr. Dhananjoi of Kirloskar presided. Dr. S. Srinivasan spoke on Surgical Emergencies, Dr. P. S. Subba Rao spoke on Modern Trends in E.N.T. Surgery, Dr. H. S. Dakshminarayana spoke on Corticosteroids, Dr. M. C. Marulappa initiated discussion on a rare and interesting case of Infantile Paralysis. Several films were shown and a variety entertainment was arranged.

COCHIN BRANCH—The general body meeting of the branch was held at Ernakulam on 7-1-61. Office-bearers for 1961-62 were elected with Dr. S. S. Rao as president, Dr. C. M. Joseph as hony. secretary; Dr. K. G. Thangamma, Dr. Vasava Menon and Dr. M. Mary Antony

as vice-presidents ; Dr. N. M. Mathai and Dr. Mrs. Isaac as joint secretaries ; Dr. C. M. Joseph as Central Council representative; Dr. Mohamed Basu as State Council representative. Dr. K. P. Achutha Menon as hony. treasurer.

A meeting of the branch was held on 28-1-61 at the residence of Dr. S. S. Rao, the president of the branch. The president in his preliminary address referred to the Travancore-Cochin Medical Practitioners' (Amendment) Bill, 1960. He laid stress on sec. 23 (ii) of the Travancore-Cochin Medical Practitioners Act 1953 and also sec. 26, elucidating all the relevant facts and suggesting the formation of a committee to go through the amendments in details, and the committee was formed in the meeting. He referred to the World Medical Journal and some of the Medical Associations and laid stress on public relations which most of the medical associations of the world have included as an important item in their planned programme. He also suggested to start a Chit Fund by which the members could offer the needed service to the poor. Mentioning the large scale resignations of experienced doctors from the Madras Medical Service, he pleaded for more reasonable approach to the service conditions of doctors. He also suggested the necessity of forming a committee to go through the service conditions and salaries of doctors in the Kerala State. Names also were suggested for the committee.

Dr. Mathew addressed the members on The Therapeutics of Steroids which was appreciated by all. The secretary thanked the speaker for his interesting talk.

COIMBATORE BRANCH—A combined meeting of the branch and the Clinical Association of the Government Headquarters Hospital, Coimbatore was held on 17-12-60. Ninety members were present. Dr. G. T. Gopalakrishna Naidu presided over the general session and Dr. V. Selvaraj presided over the clinical session. The members condoled the death of Dr. K. Sriramamurthy of Udumalpet, a member of the branch. Several interesting cases were demonstrated by the members of the various departments of the Headquarters Hospital. This was followed by a film show on Piles and the Early Detection of Rectal Cancer.

A monthly meeting of the branch was held on the 4th February 1961. Seventy members were present. Dr. D. Sundareswaran presided.

Dr. N. S. Ramamoorthy, Associate Secretary, read the minutes of the combined meeting of the Coimbatore District Medical Association and the Clinical Association of the Government Head Quarters Hospital on 17-12-60.

Dr. Jeyakumar, Madurai Medical College, then gave a very interesting and elaborate lecture on "Changing Concepts of Medicine".

The Associate Secretary, Dr. N. S. Ramamoorthy, thanked the Speaker; the hosts, Dr. S. R. Srikantam and Dr. (Mrs.) R. Saradambal and the members present.

CUTTACK BRANCH—The annual meeting of the branch was held on 21-12-60. Fifty-two members were present. Dr. G. Pattanaik presided. Office-bearers for 1960-61 were elected with Dr. G. Pattanaik as president; Dr. S. C. Misra and Dr. S. Mukherjee as vice-presidents; Dr. Nirupama Rath as secretary; Dr. R. K. Parida and Dr. U. P. Naik as joint secretaries.

DARBHANGA BRANCH—A general meeting of the branch took place on 7-2-61 in the District Board Dispensary, Sakri, under the presidentship of Dr. K. L. Misra to condole the death of Dr. S. K. Sinha, Chief Minister. A two-minutes silence was observed as a mark of respect.

DIBRUGARH BRANCH—A special meeting of the branch was held on 16-2-61 with Dr. C. R. Dutta in the chair. The members expressed their deep sense of sorrow at the sad death of Dr. U. P. Gupta, Professor of O. G. Department, Assam Medical College, Dibrugarh at Calcutta and of Dr. B. C. Sharma at Jorhat on the 4th and 14th February 1961 respectively. Both of them were members of the branch.

DOHAD BRANCH—The branch with the help of the G. S. and Kutch Territorial Branch in collaboration with the Lions' Club of Ahmedabad held one-day vasectomy camp at Dohad Municipal Hospital. Thirty-three operations were done successfully. Dr. T. B. Patel, Director of Health Services of Gujarat State, inaugurated the camp.

ETAH BRANCH –A clinical meeting of the branch was held on 5-2-61 in Civil Surgeon's Office. Dr. H. P. Banerji presided. All the local members were present. Dr. M. C. Sharma demonstrated a case of 'croup'. Besides, other cases of clinical interest were also discussed. Dr. H. P. Banerji, Civil Surgeon entertained all the members of the branch to dinner the same day.

GAYA BRANCH –An Anti-leprosy Day was observed at Gaya on 30-1-61. The committee for its observance chalked out a programme. The day began with prabhat pherries. At 10 A.M. the members of the committee paid a visit to the K. E. M. Leprosy Asylum and encouraged the patients to continue treatment. A public meeting was held in the evening in the Jawahar Town Hall with Sardar Latifur Rahman in the chair. Dr. R. Hussain spoke on Diagnosis and Treatment of Leprosy, Dr. S. N. Sinha on Rehabilitation of the Cured and Arrested Cases of Leprosy. The District Public Relations Officer gave a popular lecture stressiing on the need of co-operation of the general public. Film shows on Health were held.

GIRIDIH BRANCH—A meeting of the branch was held on 15-2-61. Dr. H. Chatterji presided.

A condolence resolution was passed on the sad demise of Late Dr. S. K. Sinha, Chief Minister of Bihar.

It was resolved to celebrate the Bihar Health Week in this sub-division from 19th to 25th March 1961. The town of Giridih with adjoining areas of the colliery and Dumri Block were especially selected for a week long health drive, health education and mass inoculation etc. A medical conference of doctors was also proposed to be held at Dumri.

JALPAIGURI BRANCH—The annual general meeting of the branch was held on 28-1-61. Twenty-seven members were present. Dr. M. K. Moitra presided. The annual report of 1959-60 was adopted. Membership showed slight increase. Total number of meetings held during the year was 22, including the working committee meetings. The activities of the branch included discussion of clinical subjects in the meetings. During the year, Dr. D. C. Talukdar of Kanchrapara T. B. Hospital spoke on Modern Treatment of Tuberculosis, (2) Dr. N. N. Sen of Kumudsankar Ray T. B. Hospital spoke on Hospital Management of Tuberculous patients, (3) Dr. M. N. Sarkar of Calcutta spoke about the present relationship between the specialists and the G.P., (4) Dr. A. K. Ghose spoke on Ocular Manifestations in Diabetes, (5) Dr. M. K. Moitra spoke on How to be a Successful Practitioner.

JORHAT BRANCH—An emergent meeting of the branch was held on 14-2-61. Dr. J. K. Saikia presided. The members expressed their deep sense of sorrow at the sudden death of Dr. B. C. Sharma, retired assistant surgeon and conveyed heartfelt condolence to the bereaved family.

KANDI BRANCH—A meeting of the branch was held on 27-11-60. Dr. S. K. Mukherjee read a paper on Acute Abdomen and its Management. Dr. S. R. Das Gupta, Chief Medical Officer, Murshidabad, read a paper on Eye Strain and Visual Hygiene at a meeting held on 14-1-61.

KARIMNAGAR BRANCH—The monthly clinical meeting was held on 22-1-61 at the District Headquarters Hospital with Dr. Shah Nawaz, late Professor of Medicine, Osmania General Hospital, Hyderabad in the chair. Dr. S. Sarfaraz Khan of Headquarters Hospital spoke on Sleepy Look and Dr. Shah Nawaz dealt with the Neurological Aspect of the Sleepy Look. Dr. Prandham of Headquarters Hospital spoke on Cerebrovascular Emergencies and Dr. Shah Nawaz gave a detailed account of

the condition and gave his experience in treating a case of Coma due to Cerebral Malaria. Dr. Ahmed Hussain, Superintendent and D.M.O., presented a case of Intestinal Obstruction recovering completely after operation.

KATIHAR BRANCH—In a meeting held on 9-1-61, the members of the branch condoled the sad and sudden death of Dr. (Mrs.) Tilotoma Prosad, a member of the branch and conveyed heartfelt sympathy to the members of the bereaved family.

KISHANGANJ BRANCH—A meeting of the branch was held on 8-2-61. Ten members were present. Dr. B. K. Das presided. The members condoled the death of Dr. S. K. Sinha, the chief minister of Bihar State. The members were requested to attend the District Medical Conferenc at Katihar.

LUCKNOW BRANCH—A meeting of the branch was held on 18-12-60 to celebrate the Family Planning Day. A symposium on Family Planning was held on the occasion. The participants were Dr. B. Mukerji, Director of Central Drug Research Institute, Dr. S. C. Misra, Prof. and Head of the Dept. of Surgery, K. G. Medical College, Prof. Sushil Chandra, Head of the Department of Sociology and Social Work, Lucknow University, Dr. Lucie Sanan Galantonio, a medical psychiatrist of Italy on UNESCO and WHO mission touring India, Dr. (Mrs.) S. L. Talwar, Asst. Director, Family Planning, U.P., Mrs. V. Kercood, Dy. Director, Social Welfare, Dr. K. M. Lal, Director of Medical and Health Services, U.P., Dr. M. V. Gujral, Principal and Head of the Dept. of Pharmacology, K. G. Medical College. The symposium was followed by film shows on the subject. Keen interest was evinced by the public.

MADHIPURA BRANCH—An ordinary meeting of the branch was held on 16-1-61. Six members were present. Dr. R. P. Singh was re-elected president of the branch. Dr. H. N. Das also was re-elected as secretary for 1960-61. A committee was formed to celebrate the Health Week and the president was requested to organise the function from 5-3-61 to 12-3-61. The Special officer, District Board, Saharsa, and the Presidency Notified Area Committee, Madhipura, were requested to allot Rs. 75/- and Rs. 50/- respectively to meet the expenses of the Health Week celebration.

A special meeting of the branch held on 4-2-61 recorded its deep sense of sorrow at the death of Dr. Srikrishna Sinha, the chief minister of Bihar. Two minutes' silence was observed as a mark of respect.

Dr. H. Das, the secretary of the branch, read a short life sketch of late Dr. Srikrishna Sinha at the meeting.

MADRAS CITY BRANCH—A meeting of the Executive Committee of the branch was held on 8-11-60 at Hotel Dasaprakash. Dr. G. Sriramulu presided. Ten members were present. It was decided that resolutions which get specific direction from the committee can be sent before confirmation by the subsequent meeting. Dr. P. Alagasingari Naidu and Dr. R. Sankaran were re-instated. Twenty-five new members were admitted. Dr. A. M. Sivaraman pointed out that a meeting should be arranged when Dr. Balasundaram and Dr. Mehta be requested to speak about their experiences of their recent I.M.A. European tour.

A clinical meeting was held on 5-11-60. Dr. Sriramulu presided. Dr. E. Dunlop, Hunterian Professor of the Royal College of Surgeons, England and Cecil Joll Medallist of the Royal College delivered a lecture on the Problems of the Mouth and Throat Cancer. He gave an authoritative exposition of cancer surgery in mouth and throat with slides, and advocated radiotherapy only when cancer surgeons failed to give satisfactory results.

A meeting of the executive committee was held on 6-12-60. Dr. G. Sriramulu presided. Thirteen members were present. The members by a resolution requested the government to sanction free expenditure for purchasing an audio-visual apparatus namely combined projector and epidiascope. Twenty new members were admitted. Two alternative sites were suggested for the building, for applying to the Collectorate of Madras for allotment. The letter of Dr. H. N. Shivapuri requesting the branch to hold the All-India Medical Conference in March was considered. The branch regretted its inability.

MADURA BRANCH—The monthly meeting of the branch was held on 17-12-60. Dr. R. S. Chellappa presided. Dr. M. D. Ananathachari, late Principal, Madurai Medical College, gave a lecture on the Problem of Amoebiasis.

The monthly meeting was held on 21-1-61. Dr. K. Ramachandran presided. Condolence resolutions on the death of Dr. M. N. Venkataraman and Dr. B. R. Venkatasubramaniam were passed. It was decided to convey the resolutions to the bereaved families. Dr. G. Venkataswamy of Madurai Medical College, spoke on Eye Examination in General Diseases.

MANDVI BRANCH—A clinical meeting was held on 1-2-61. Dr. V. P. Dandakar presided. Dr. P. O. Trivedi of Maska T. B. Clinic presented a few cases of various lung conditions. The cases were demonstrated with the help of skiagrams. Dr. C. J. Mehta also demonstrated a case of Bronchial Asthma which turned out to be a case of tuberculosis, the skiagram having the unusual appearance of Eosinophilic Lung. Dr. Dandakar said that unless and until the socio-economic conditions of the state are improved it was difficult to check the spread of this disease.

MEDAK BRANCH—The monthly clinical meeting of the branch was held on 12-2-61. Dr. Ibrahim Ali Khan presided. Dr. V. Raghvender Rao, hony. secretary, requested the members to attend the IV Andhra Pradesh Medical Conference at Kakinada on 26-27 February 1961. Dr. Muneeruddin presented a case of Hypertension. Dr. B. Reddy spoke on a case of Ectopic Gestation and a case of Blast Injury. Dr. M. A. Aziz read a paper on Pitfalls in the Diagnosis of Hydrocele.

MIDNAPUR BRANCH—The branch observed anti-leprosy day on 30-11-61. A public meeting was organised in the evening with Prof. S. S. Bari, Chairman, Midnapur Municipality. Dr. A. B. Sen, hony. secretary, I.M.A., Midnapur, gave an outline of the incidence of leprosy in Midnapur. Sri S. R. Dutt, Sri J. C. Das, Dr. G. C. Pal, Dr. M. Das and Sri S. Banerjee discussed the leprosy problem in Midnapur town and its control. Dr. M. Das gave a detailed account of leprosy, regarding epidemiology, methods of control, etc.

NAGAPATTINAM BRANCH—A meeting of the branch was held on 18-2-61 at the T.B. Association Buildings. When Dr. A. Thiyagarajon, Retd. D.M.O. presided and Dr. N. Srinivasan spoke on the "Infections of the Hand" with special reference to mill workers.

NIZAMABAD BRANCH—A meeting of the branch was held on 12-1-61. Dr. Mohd. Gulam Nabi, District Medical Officer, presided. Dr. Razzack presented a case of Brain Tumour in a Child. Dr. Gulam Nabi presented a case of Cancer Pylorus in a Young Male.

PRATAPGARH BRANCH—To observe Anti-Leprosy Day a meeting of the branch was held on 30-1-61 at the residence of Dr. Lajpat Rai Varma, Malaria Eradication Officer, Pratapgarh. Dr. H. M. Kar, Civil Surgeon, Pratapgarh, presided.

Dr. H. M. Kar spoke on Treatment of Leprosy. Dr. Mukherjee spoke on Aetiology, Diagnosis and regional aspect of the disease as it prevails in various parts of India at present. Socio-Medical and Preventive measures were also discussed.

PILIBHIT BRANCH—The members of the branch mourned the sad and untimely death of Dr. Y. P. Seth, Municipal Medical Officer of Health, Pilibhit at the age of 37 years and conveyed their heart-felt sympathy to the members of the bereaved family.

RANCHI BRANCH—At the annual general meeting, office-bearers for 1960-61 were elected with Dr. L. P. Verma as president; Dr. Ramanaj Singh as vice-president; Dr. B. N. Roy as secretary; Dr. A. R. N. Prasad as joint secretary; Dr. N. P. Srivastava as treasurer; Dr. S. S. Sahay as auditor.

ROORKEE BRANCH—A meeting was held on 31-12-60. Dr. R. C. Srivastava presided. Office-bearers for 1960-61 were elected with Dr. R. C. Srivastava as president; Dr. B. C. Mazumdar as secretary; Dr. Sambhu Nath as treasurer. A paper on Diabetes was read by Dr. R. C. Srivastava. The members accepted the invitation of the O. C. Military Hospital to hold the next meeting there on 30-1-61.

ROURKELA BRANCH—The annual day of the branch was held on 20-11-60. Dr. S. K. Ghosh presented the annual report for 1959-60 in which he gave a summary of the activities of the branch. Office-bearers for 1960-61 were elected with Dr. G. K. Bergter as president; Dr. B. K. Das as secretary; Dr. A. K. Mitra as treasurer. A symposium on Diabetes Mellitus was held. Dr. B. S. Dhillon, Chief Medical Officer of Rourkela Steel Project presided. Dr. J. B. Mitra spoke on Introduction and History; Dr. A. K. Mitra on Metabolic Disorder and Physiological Aspects of Diabetes; Dr. J. N. Bhatta on Pathology; Dr. J. Pati on Medical Complications; Dr. K. C. Mishra on Treatment and Therapeutics; Dr. R. Singh Deo on Juvenile Diabetes and its Role in Heredity; Dr. A. Roy on Diabetes as a Problem to the Surgeon; Dr. T. K. Ghosh on Ocular Complications; Dr. D. P. Dey on Pregnancy in Diabetes; Dr. B. K. Das on Dermatological Manifestations; Dr. V. V. Deshmukh on Anaesthesia in Diabetes; Dr. S. K. Ghosh on Role of Roentgenology in Diabetes Mellitus.

SAKRI BRANCH—A general meeting of the branch took place on 7-2-61. Dr. K. L. Misra presided. The members condoled the death of Dr. S. K. Sinha, Chief Minister, Bihar. Two minutes' silence was observed as a mark of respect.

A meeting of the branch was held on 30-10-60. Dr. S. M. Razi presided. Dr. B. N. Das Gupta of Darbhanga Medical College attended as a guest. Dr. B. N. Das Gupta gave a refresher course lecture on Pitfalls in Medical Diagnosis.

SHILLONG BRANCH—A clinical cum general meeting was held on 4-2-61 at Pasteur Institute, Shillong. Dr. S. Bhattacharya presided. Thirty-two members were present. Dr. (Miss) N. Chaliha of Ganeshdas Hospital spoke on Menorrhagia, Dr. K. Joshi and Dr. J. Tariang of Ganeshdas Hospital presented 2 case notes which were demonstrated. Case 1 was diagnosed as a case of Cirrhosis of Liver and the second one, a case of Acute Nephritis and Splenic Anaemia. It was decided to hold the annual general meeting in the 3rd week of March, 1961. Dr. (Miss) Chaliha was elected a member of the Central Council, and Dr. S. Bhattacharya, Dr. S. Chaudhuri, Dr. C. N. Hazarika, Dr. (Miss) N. Chaliha, Dr. L. M. Kakati, Dr. S. C. Deb, Dr. A. Biswas and Dr. N. Das were elected to the State Council. A sub-committee was formed to meet the government to realise the Government contributory Health Scheme, to explain the difficulties of the existing medical practitioners and to request the government to utilise their services.

SIBSAGAR BRANCH—The members of the branch in a meeting held on 1-12-60 condoled the death of Dr. U. C. Bordoloi, a member of I.M.A., retired D.H.S., Assam, a renowned gynaecologist.

SINDRI BRANCH—The annual general meeting of the branch was held on 22-1-61. Dr. B. C. Aich, Chief Medical Officer of the Fertilizer Corporation of India Ltd., Sindri, presided. Office-bearers for 1960-61 were elected with Dr. B. C. Aich as president; Dr. N. Vyathanathan and Dr. B. S. Dutt as vice-presidents; Dr. L. P. Chowdhury as hony. secretary; Dr. B. N. Choudhury as financial secretary; Dr. L. P. Choudhury as a member of the State Council. The annual report for 1959-60 showed an increase of membership by 2. Eight meetings were held in 1959-60 and 3 clinical meetings were also held. Bihar Health Week was observed during the year.

TANGLA BRANCH—A meeting of this branch was held on 20-11-60 with Dr. K. C. Chakravarty as president. Ten members were present. Office-bearers for the year 1961-62 were elected as follows. Dr. B. M. Chakraborty as president and Dr. T. P. Chaudhury as hony. secretary. A paper on Dysentery in Tea Estates was read by Dr. T. P. Chaudhury which was appreciated.

TIRUCHY BRANCH—A monthly meeting of the branch was held on 17-12-60. Dr. P. Sundaram presided. Forty members were present. At the outset the members condoled the death of Dr. D'Costa, an old lady doctor of Tiruchi. Dr. M. V. Bhat, Professor of Operative Surgery, Madurai Medical College, gave a talk on Haematuria, its Cause and Treatment.

UNNAO BRANCH—Office-bearers of the branch for 1960-61 were elected with Dr. K. D. Jain, Civil Surgeon as president; Dr. R. Sahi as vice-president; Dr. S. S. Tewari as hony. secretary; Dr. D. D. Talwar as auditor and joint secretary; Dr. S. R. Chakraborty as representative for Central Council; Dr. A. C. Bhatnagar as representative for State Council.

U. P. STATE BRANCH—The 2nd meeting of the Working Committee of the State Branch was held on 22-12-60. Twelve members were present. The members condoled the death of Dr. K. P. Roy Chowdhury (Varanasi) and Dr. Raghunandan Lal (Lucknow). The proceedings of the last meeting were confirmed. The audited accounts for 1959-60 were confirmed. It was decided to present a Replica of the Insignia to each retiring president and money was sanctioned for preparing 4 Replicas of the Insignia to be presented to the 4 past presidents. A sub-committee was formed in connection with the Government notification regarding permit for drugs, to submit a memorandum to the Government and if necessary to meet the minister. A sub-committee was formed with Dr. R. N. Gupta and Dr. Kedar Nath with power to co-opt, to submit a detailed scheme about reorganising the scientific section of the annual conference at State level. Regarding Dr. C. M. Aga's letter, it was decided to request the Director of Medical and Health Services, U. P., that the cases admitted to district hospitals through the members of the profession should be referred back to them at the time of discharge of the patients with short notes about the case. The working committee accepted the invitation of the Lucknow branch to hold the 26th U. P. State Medical Conference at Lucknow. The secretary suggested that each member should have an index card which will be in a way, the life-history of the member. The suggestion of Dr. H. N. Shivapuri that the secretary should collect information and reply to the questionnaires on Rural Health Services was accepted. On the suggestion of Dr. Bholanath, the secretary was asked to write a letter of protest to the Chief Minister, U. P., regarding his speech at a meeting of Ayurveds.

VISHAKHAPATNAM BRANCH—The monthly general body meeting of the branch was held at King George Hospital on 28-1-61. Dr. S. Balaparameswararao, Neurosurgeon, K. G. Hospital, spoke about his experiences of his stay in U. K. for 18 months and recent developments in the various fields of neuro-surgery and neuro-pathology, etc.

XIV GUJARAT, SAURASHTRA AND KUTCH TERRITORIAL MEDICAL CONFERENCE, 1961, RAJKOT

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Further information may be obtained from Dr. Trilokbhai M. Shah, hony. branch secretary, Sir Lakhhji Raj Road, Rajkot (Saurashtra).

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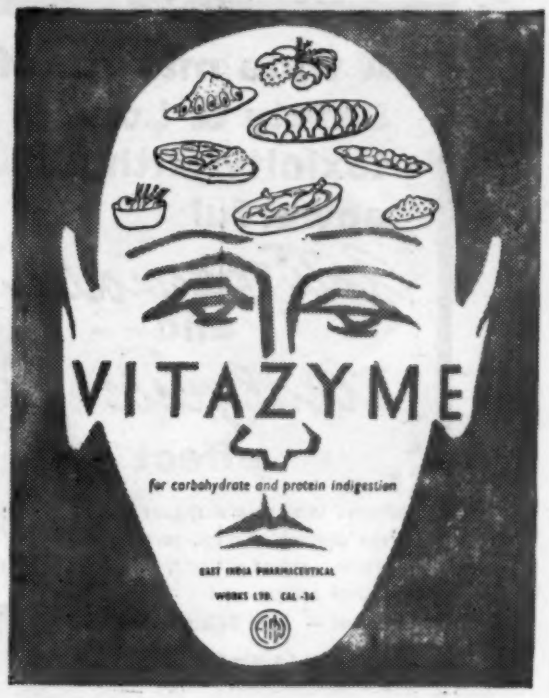

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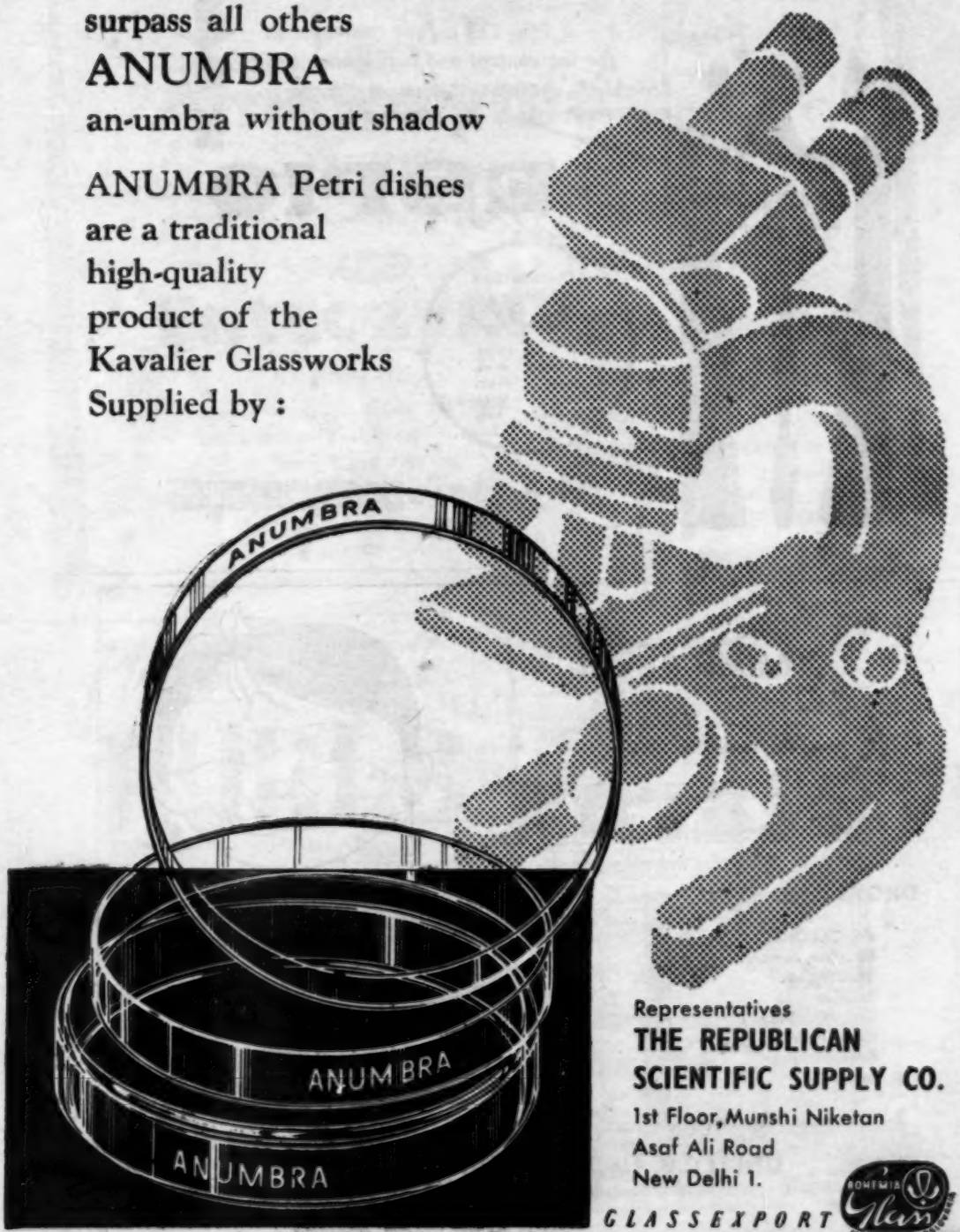

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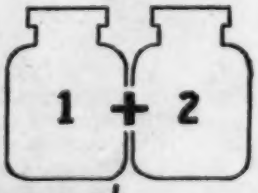

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